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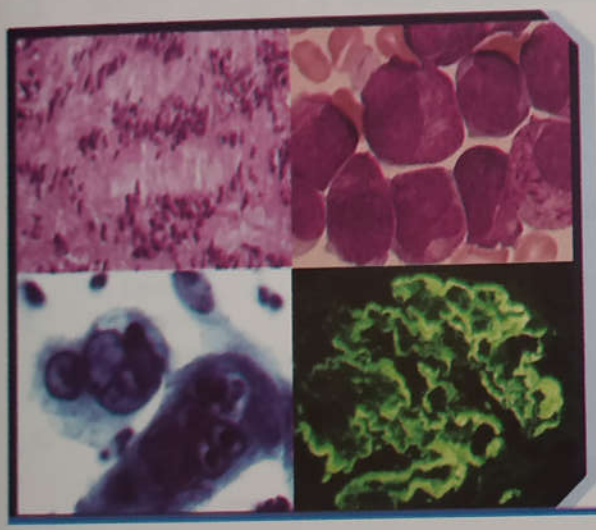

Pathology

Cell as a Unit of Health and Disease – Questions

1. **Which of the following plays a role in gene editing?**
   a. Gene Xpert
   b. CRISPR
   c. Heath care apps
   d. Big data

2. **Cell to cell permeability occurs through:**
   a. Occludin
   b. Zona adherens
   c. Connexins
   d. Zonulin

3. **Which Collagen is typical of basement membrane?**
   a. Type I
   b. Type II
   c. Type III
   d. Type IV

4. **All of the following are Intermediate filament except?**
   a. Lamin
   b. Cadherin
   c. Vimentin
   d. Desmin

5. **Titin protein mutated in?**
   a. DCM
   b. HOCM
   c. RCM
   d. Non-functional cardiomyopathy

6. **Bridging fibrosis in large wounds is due to:**
   a. Keratinocyte growth factor
   b. Epidermal growth factor
   c. Platelet derived growth factor
   d. Transforming growth factor-β

7. **Types of collage playing important role in wound healing**
   a. I and III
   b. II and V
   c. III and IV
   d. V and IX

8. **Oval stem cells are located in-**
   a. Canal of schlemm
   b. Canal of herring
   c. Space of disse
   d. Basal lamina of myotubules

9. **Heritable changes in gene expression not caused by alterations in DNA sequence refers to?**
   a. Genetics
   b. Epigenetics
   c. Mutations
   d. Transposons

10. **Epigenetic factors refers to?**
    a. Histone methylation
    b. Histone phosphorylation
    c. DNA methylation
    d. All of the above

11. **Collagen present in cornea?**
    a. Type 1
    b. Type 2
    c. Type 4
    d. Type 7

12. **Gene silencing RNA-**
    a. rRNA
    b. tRNA
    c. miRNA
    d. None

13. **The most abundant glycoprotein present in basement membrane is-**
    a. Laminin
    b. Fibronectin
    c. Collagen type 4
    d. Heparan sulphate

1. b	2. c	3. d	4. b	5. a	6. d	7. a	8. b	9. b	10. d	11. b
12. c	13. a									

14. Which of the following statement is not true?

a. Cytokeratin is the marker of muscle cells
b. Vimentin is used to stain fibroblasts
c. Neurofilaments are present in neurons
d. Desmin can be used as a marker for muscle cells

15. Which of the following is both morphogenic and mitogenic?

a. Insulin growth factor
b. Bone morphogenic factor
c. Fibroblast growth factor
d. Epidermal growth factor

16. Which of the following is involved in stem cell self renewal and pluripotency?

a. Oct 3/4
b. sox 2
c. c-myc
d. FLT3 ligand
e. c-kit

17. All of the following are true about single nucleotide polymorphisms except

a. Almost always biallelic
b. 1% occur in non coding regions
c. Can be neutral variant with no effect on gene function
d. Can serve as a marker for disease

18. Which of the following statements is true?

a. Actin is the most abundant cytosolic protein in the cell
b. Lamin is the most abundant glycoprotein in the basement membrane
c. Fibronectin imparts scaffolding to the cytoplasm
d. FACITs are seen in association with type I collagen

19. Which of the following pairs of cell cycle phase and cyclin/CDK is incorrectly matched?

a. G1S checkpoint- cyclin D/CDK 6
b. G1S checkpoint- cyclin D/CDK4
c. S phase- cyclin A/CDK 6
d. G2M checkpoint- cyclin B/CDK1

20. Which of the following organ and site of stem cell is incorrectly matched?

a. Liver- oval cells in Space of Disse
b. Skin- bulge of hair follicle
c. Brain- dentate gyrus
d. Cornea- limbus

21. Major cytokine involved in fibrosis

a. Transforming growth factor-α
b. Transforming growth factor-β
c. Fibroblast growth factor
d. Epidermal growth factor

22. Most abundant collagen in the body?

a. Type I
b. Type II
c. Type III
d. Type IV

23. FACIT collagen is?

a. Type I
b. Type III
c. Type IX
d. Type XI

14. a	15. b	16. a, b, c, e	17. b	18. a	19. c	20. a	21. b	22. a	23. c

Cell as a Unit of Health and Disease – Explanations

1. B

Reference: Robbins Basic Pathology, 10th Edition

Clustered regularly interspaced short palindromic repeats (CRISPRs) and Cas (or CRISPRassociated genes) are linked genetic elements that endow prokaryotes with a form of acquired immunity to phages and plasmids. Bacteria use this system to sample the DNA of infecting agents, incorporating it into the host genome as CRISPRs. CRISPRs are transcribed and processed into an RNA sequence that binds and directs the nuclease Cas9 to a sequences (e.g., a phage), leading to its cleavage and the destruction of the phage. Gene editing repurposes this process by using artificial guide RNAs (gRNAs) that bind Cas9 and are complementary to a DNA sequence of interest. Once directed to the target sequence by the gRNA, Cas9 induces double-strand DNA breaks.

Option A- The Xpert MTB/RIF is a cartridge-based nucleic acid amplification test for simultaneous rapid tuberculosis diagnosis and rapid antibiotic sensitivity test. It is an automated diagnostic test that can identify Mycobacterium tuberculosis DNA and resistance to rifampicin

Option C and Option D are far removed from gene editing and have no bearing whatsoever to Genomics

2. C

Reference: Robbins Pathological Basis of Disease, 9th edition

Communicating junctions (gap junctions) mediate the passage of chemical or electrical signals from one cell to another. The junction consists of a dense planar array of 1.5- to 2-nm pores (called connexons) formed by hexamers of transmembrane protein connexins. These pores permit the passage of ions, nucleotides, sugars, amino acids, vitamins, and other small molecules; the permeability of the junction is rapidly reduced by lowered intracellular pH or increased intracellular calcium.

Option A and D- Occluding junctions (tight junctions) seal adjacent cells together to create a continuous barrier that restricts the paracellular (between cells) movement of ions and other molecules. Viewed en face, occluding junctions form a tight meshlike network of macromolecular contacts between neighboring cells. The complexes that mediate these cell–cell interactions are composed of multiple proteins, including occludin, claudin, zonulin, and catenin

Option B- When the adhesion focus is between cells, and is small and rivet-like, it is designated a spot desmosome or macula adherens. When such a focus attaches the cell to the ECM, it is called a hemidesmosome. Similar adhesion domains can also occur as broad bands between cells, where they are denoted as belt desmosomes or zona adherens.

3. D

Reference: Robbins Pathological Basis of Disease, 9th edition

Non-fibrillar collagens may contribute to the structures of planar basement membranes (type IV collagen).

4. B

Intermediate filaments are 10-nm diameter fibrils that comprise a large and heterogeneous family. Individual types have characteristic tissue-specific patterns of expression that can be useful for assigning a cell of origin for poorly differentiated tumors.

- Lamin A, B, and C: nuclear lamina of all cells
- Vimentin: mesenchymal cells (fibroblasts, endothelium)
- Desmin: muscle cells, forming the scaffold on which actin and myosin contract

- Neurofilaments: axons of neurons, imparting strength and rigidity
- Glial fibrillary acidic protein: glial cells around neurons
- Cytokeratins: at least 30 distinct varieties, subdivided into acidic (type I) and neutral/basic (type II); different types present in different cells, hence can be used as cell markers

Option A and D- Intermediate filaments

Option B- Cell-cell desmosomal junctions are formed by homotypic association of transmembrane glycoproteins called cadherins

5. A

Reference: Robbins Pathological Basis of Disease, 9th edition

DCM is familial in at least 30% to 50% of cases, in which it is caused by mutations in a diverse group of more than 20 genes encoding proteins involved in the cytoskeleton, sarcolemma, and nuclear envelope (laminin A/C). In particular, mutations in TTN, a gene that encodes titin (so-called because it is the largest protein expressed in humans), may account for approximately 20% of all cases of DCM.

Option B- Mutations causing HCM are found most commonly in the gene encoding β-myosin heavy chain (β-MHC), followed by the genes coding for cardiac TnT, α-tropomyosin, and myosin-binding protein C (MYBP-C); overall, these account for 70% to 80% of all cases.

Option C- Restrictive c
be idiopathic or assoc
diseases or processes t
cardium, principally rac
yloidosis, sarcoidosis,
or the deposition of me
mulate due to inborn er

Option D- It is a non sp

6. D

Reference: Robbins Pa
Disease, 9th edition

TGF-β drives scar
applies brakes on the
accompanies wound he

- TGF-β stimulates
collagen, fibronectin,
and it inhibits collag
both decreasing matri
(MMP) activity and inc
of tissue inhibitors of
is involved not only
after injury, but also
lung, liver, and kidne
chronic inflammation
- TGF-β is an antiinf
that serves to lin
inflammatory respon
inhibiting lymphocyt
the activity of other
models lacking TGF-
and persistent inflam

Option A, B and C- Re
table

Growth Factor	Sources	Func
Epidermal. growth factor (EGF)	Activated macrophages, salivary glands, keratinocytes, and many other cells	Mitogenic for keratinocyt stimulates keratinocyte m formation of granulation
Transforming growth factor-α (FGF-α)	Activated macrophages, keratinocytes, many other cell types	Stimulates proliferation o other epithelial cells
Hepatocyte growth factor (HGF) (scatter factor)	Fibroblasts, stromal cells In the liver, endothelial cells	Enhances proliferation of epithelial cells; Increases
Vascular endothelial growth factor (VEGF}	Mesenchyme cells	Stimulates proliferation Increases vascular perme

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Platelet-derived growth factor IPOGF)	Platelets, macrophages, endothelial cells, smooth muscle cells, keratinocytes	Chemotactic for neutrophils, macrophages, fibroblasts, and smooth muscle cells; activates and stimulates proliferation of fibroblasts, endothelial, and other cells; stimulates ECM protein synthesis
Fibroblast growth factors (FGFs), Including acidic (FGF-1) and basic (FGF-2)	Macrophages, mast cells, endothelial cells, many other cell types	Chemotactic and mitogenic for fibroblasts; stimulates angiogenesis and ECM protein synthesis
Transforming growth factor-β (TGF-β)	Platelets, T lymphocytes, macrophages, endothelial cells, keratinocytes, smooth muscle cells, fibroblasts	Chemotactic for leukocytes and fibroblasts; stimulates ECM protein synthesis; suppresses acute Inflammation
Keratinocyte growth factor (KGF) (i.e., FGF-7)	fibroblasts	Stimulates keratinocyte migration, proliferation, and differentiation
ECM Extracellular membrane.		

7. A

Reference: Robbins Pathological Basis of Disease, 9th edition

At first a provisional matrix containing fibrin, plasma fibronectin, and type III collagen is formed, but in about 2 weeks this is replaced by a matrix composed primarily of type I collagen. Ultimately, the original granulation tissue scaffold is converted into a pale, avascular scar, composed of spindleshaped fibroblasts, dense collagen, fragments of elastic tissue, and other ECM components

8. B

Reference: Robbins Pathological Basis of Disease, 9th edition

In situations where the proliferative capacity of hepatocytes is impaired, such as after chronic liver injury or inflammation, progenitor cells in the liver contribute to repopulation. In rodents, these progenitor cells have been called oval cells because of the shape of their nuclei. Some of these progenitor cells reside in specialized niches called canals of Hering, where bile canaliculi connect with larger bile ducts. The signals that drive proliferation of progenitor cells and their differentiation into mature hepatocytes are topics of active investigation.

Option A- circular lymphatic-like vessel in the eye that collects aqueous humor from the anterior chamber and delivers it into the episcleral blood vessels via aqueous veins

Option C- location in the liver between a hepatocyte and a sinusoid. It contains the blood plasma. Microvilli of hepatocytes extend into this space, allowing proteins and other plasma components from the sinusoids to be absorbed by the hepatocytes. Fenestration and discontinuity of the endothelium, as well as its basement membrane, facilitates this transport.

Option D- Site of regeneration

9. B

Option A- Genetics is the study of genes, genetic variation, and heredity in living organism

Option C- A gene mutation is a permanent alteration in the DNA sequence

Option D- DNA sequence that can change its position within a genome, sometimes creating or reversing mutations and altering the cell's genetic identity and genome size. Transposition often results in duplication of the same genetic material.

10. D

Epigenetic factors include-

- Histone methylation
- Histone phosphorylation
- Histone acetylation
- DNA methylation
- Chromatin organising factors

11. B

COLLAGEN TYPE	TISSUE DISTRIBUTION	GENETIC DISORDERS
Fibrillar Collagens		
I	Ubiquitous in hard and soft tissues	Osteogenesis Imperfecta
II	Cartilage, intervertebral disc, vitreous	Achondrogenesis type 2
III	Hollow organs, soft tissues	Vascular Ehler Danlos Syndrome
V	Soft tissues, blood vessels	Classical Ehler Danlos Syndrome
IX	Cartilage, vitreous	Stickler Syndrome
Basement Membrane Collagen		
IV	Basement membranes	Alport Syndrome

12. C

Noncoding RNAs are encoded by genes that are transcribed but not translated. Although many distinct families of noncoding RNAs exist, we will only discuss two examples here: small RNA molecules called microRNAs, and long noncoding RNAs >200 nucleotides in length. Post transcriptional silencing of gene expression by miRNA is a fundamental and well-conserved mechanism of gene regulation present in all eukaryotes (plants and animals).

Option A- ribosomal RNA Option B- transfer RNA

13. A

Prototypical adhesive glycoproteins include fibronectin (a major component of the interstitial ECM) and laminin (a major constituent of basement membrane). Integrins are representative of the adhesion receptors, also known as cell adhesion molecules (CAMs); the CAMs also include immunoglobulins, cadherins, and selectins.

Option A- Laminin is the most abundant glycoprotein in basement membrane. It is an 820-kD cross-shaped heterotrimer that connects cells to underlying ECM components such as type IV collagen and heparan sulfate. Besides mediating attachment to basement membrane, laminin can also modulate cell proliferation, differentiation, and motility

Option B- Fibronectin is a large (450 kD) disulfide-linked heterodimer that exists in tissue and plasma forms; it is synthesized by a variety of cells, including fibroblasts, monocytes, and endothelium.

Option C- Basement membrane collagen

Option D- Proteoglycans form highly hydrated compressible gels that confer resistance to compressive forces; in joint cartilage, proteoglycans also provide a layer of lubrication between adjacent boney surfaces. Proteoglycans consist of long polysaccharides, called glycosaminoglycans (examples are keratan sulfate and chondroitin sulfate) attached to a core protein; these are then linked to a long hyaluronic acid polymer called hyaluronan, in a manner reminiscent of the bristles on a test tube brush.

14. A

Cytokeratin is a marker for epithelial cells

Option B- Vimentin is a marker for mesenchymal cells and tumors arising from them

Option C- stains axons of neurofilaments

Option D- stains muscle cells and tumors arising from them

15. B

Bone morphogenetic proteins (BMPs) are multi-functional growth factors that belong to the transforming growth factor beta (TGFbeta) superfamily and are both morphogenic and mitogenic.

Growth Factor	Sources	Functions
Epidermal. growth factor (EGF)	Activated macrophages, salivary glands, keratinocytes, and many other cells	Mitogenic for keratinocytes and fibroblasts; stimulates keratinocyte migration; stimulates formation of granulation tissue
Transforming growth factor-α (FGF-α)	Activated macrophages, keratinocytes, many other cell types	Stimulates proliferation of hepatocytes and many other epithelial cells
Hepatocyte growth factor (HGF) (scatter factor)	Fibroblasts, stromal cells In the liver, endothelial cells	Enhances proliferation of hepatocytes and other epithelial cells; Increases cell motility
Vascular endothelial growth factor (VEGF}	Mesenchyme cells	Stimulates proliferation of endothelial cells; Increases vascular permeability
Platelet-derived growth factor IPOGF)	Platelets, macrophages, endothelial cells, smooth muscle cells, keratinocytes	Chemotactic for neutrophils, macrophages, fibroblasts, and smooth muscle cells; activates and stimulates proliferation of fibroblasts, endothelial, and other cells; stimulates ECM protein synthesis
Fibroblast growth factors (FGFs), Including acidic (FGF-1) and basic (FGF-2)	Macrophages, mast cells, endothelial cells, many other cell types	Chemotactic and mitogenic for fibroblasts; stimulates angiogenesis and ECM protein synthesis
Transforming growth factor-β (TGF-β)	Platelets, T lymphocytes, macrophages, endothelial cells, keratinocytes, smooth muscle cells, fibroblasts	Chemotactic for leukocytes and fibroblasts; stimulates ECM protein synthesis; suppresses acute Inflammation
Keratinocyte growth factor (KGF) (i.e., FGF-7)	fibroblasts	Stimulates keratinocyte migration, proliferation, and differentiation

ECM Extracellular membrane.

16. A, B, C, E

Pluripotency of embryonic stem cell depends on expression of following transcription factors-

1. Oct 3/4
2. Sox 2
3. C-Myc
4. Kfl 4
5. ckit

- Pluripotency of embryonic stem cell is inhibited by home box protein 'Nanog'.

17. B

The two most common forms of DNA variation in the human genome are single-nucleotide polymorphisms (SNPs) and copy number variations (CNVs). SNPs are variants at single nucleotide positions and are almost always biallelic (i.e., only two choices exist at a given site within the population, such as A or T). Much effort has been devoted to mapping common SNPs in human populations. Over 6 million human SNPs have been identified, many of which show wide variation in frequency in different populations. SNPs occur across the genome—within exons, introns, intergenic regions, and coding regions. Overall, about 1% of SNPs occur in coding regions, which is about what would be expected by chance, since coding regions comprise about 1.5% of the genome. SNPs located in non-coding regions may fall in regulatory elements in the genome, thereby altering gene expression; in such instances the SNP may have a direct influence on disease susceptibility. In other instances, the SNP may be a "neutral" variant that has no effect on gene function or carrier phenotype. However, even "neutral" SNPs may be useful markers if they

happen to be co-inherited with a disease-associated gene as a result of physical proximity. In other words, the SNP and the causative genetic factor are in linkage disequilibrium. There is hope that groups of SNPs may serve as markers of risk for multigenic complex diseases such as type II diabetes and hypertension. However, the effect of most SNPs on disease susceptibility is weak, and it remains to be seen if identification of such variants, alone or in combination, can be used to develop effective strategies for disease prevention

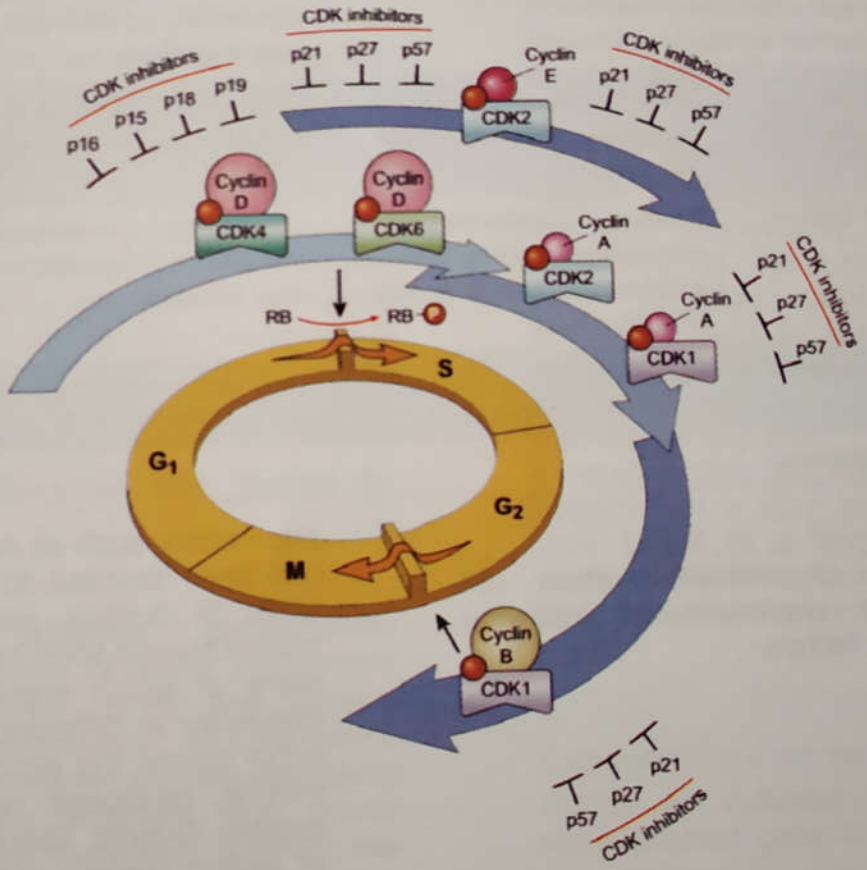
18. A

Laminin is the most abundant glycoprotein. Lamin is an intermediate filament of the cytosol.

Fibronectin is a component of the extra cellular matrix, not cytoplasm.

FACITs (fibril-associated collagen with interrupted triple helices), such as type IX collagen are in cartilage.

19. C



20. A

Tissue and Stem Cells	Location	Extra
Bone marrow= HSCs and MSCs	HSCs can be collected from bone marrow, umbilical cord blood and peripheral blood of individuals receiving CSF's	Bone marrow produces 1.5 x 106 blood cells per second
Liver- Oval cells (Bipotential-hepatocytes or biliary cells)	Canal of Hering- junction between the biliary ductal system and parenchymal hepatocytes	Activated only when hepatocyte proliferation is stopped

Brain- Neural Stem Cells (NSCs)	Subventricular zone and Dentate gyrus of Hippocampus	Capable of generating neurons, astrocytes and oligodendrocytes
Skin	Hair Follicle Bulge Interfollicular area of surface epidermis Sebaceous glands	Activated by Wnt pathway and inhibition of signalling from BMP pathway
Intestine	Above paneth cells in SI and base of crypt in case of colon	Wnt and BMP pathways
Skeletal Muscle	Beneath myocyte basal lamina	Regeneration is through Satellite cell replication
Cornea	At the junction between the epithelium of cornea and conjunctiva	Limbal Stem Cells (LSCs)

21. B

22. A

23. C

Cell Adaptation, Injury and Death – Questions

24. True about Metaplasia is?

a. Involves only epithelial cells
b. Is irreversible
c. Can occurs at stem cells level
d. Columnar is the most common type

25. All of the following true regarding hypertrophy except:

a. Increased in cell size without increase in number
b. DNA content same as in normal cells
c. Increase in cell size is due to synthesize of more cellular proteins
d. Associated with a switch of contractile proteins from adult to fetal or neonatal forms

26. Find the false statement about metaplasia:

a. Reversible
b. No loss of polarity
c. Reprogramming of stem cells
d. Pleomorphism present

27. Increase in the number of goblet cells in the non-respiratory terminal bronchiole is an example of:

a. Anaplasia
b. Dysplasia
c. Metaplasia
d. Hyperplasia

28. In respiratory tract metaplasia occurs from:

a. Squamous to columnar
b. Columnar to cuboidal
c. Columnar to squamous
d. Cuboidal to squamous

29. In Vitamin-A deficiency, cancerous lesions occur due to-

a. Metaplasia
b. Dysplasia
c. Aplasia
d. Hyperplasia

30. All are cellular adaptations except:

a. Hypertrophy
b. Hyperplasia
c. Necrosis
d. Metaplasia

31. Features of irreversible cell injury is/are? (PGI type)

a. Lysosomal rupture
b. Pyknosis
c. Bleb formation on membrane
d. Severe mitochondrial dysfunction

32. In myocardium reperfusion injury is due to?

a. Ca
b. Mg
c. K
d. Mn

33. Which of the following is not a sign of reversible cell injury?

a. ATP depletion
b. Cell shrinkage
c. Fatty acid deposition
d. Reduction of phosphorylation

34. Cells seen in chronic infection of pseudomonas:

a. Neutrophils
b. Eosinophils
c. Lymphocytes
d. Macrophage

35. For programmed cell death and autopagy, which is pro apoptotic genes?

a. BCL2
b. BAX
c. BCL-XL
d. BIM

24. c	25. b	26. d	27. c	28. c	29. a	30. c	31. a,d	32. a	33. b	34. a
35. b										

36. True about p53 gene? (PGI type)
a. Tumor suppression
b. Proapoptotic
c. Antiapoptotic
d. Cell repair
e. Oncogene

37. True about caspases are?
a. They are enzymes starting apoptosis
b. They inhibit apoptosis
c. They are receptors of apoptosis
d. They are proteases which cause cellular death in apoptosis

38. True about necroptosis is all except?
a. Caspase 1 & 11 is involved
b. Caspase independent
c. Failure of activation of caspase 8
d. Lipid peroxidation is seen

39. SMAC/DIAMBLO is a:
a. Anti apoptotic protein
b. Induces necrosis
c. Acts both as anti and pro apoptotic protein
d. Pro-apoptotic protein

40. Which of the following is an execution caspase:
a. Caspase 3
b. Caspase 5
c. Caspase 8
d. Caspase 9

41. Annexin V is a marker of-
a. Necrosis
b. Gangrene
c. Aging
d. Apoptosis

42. The following is not a sensor of apoptosis:
a. Puma b. Noxa
c. BAX d. BAD

43. Intrinsic pathway of apoptosis is initiated by all the following except:
a. Growth factor withdrawal
b. DNA damage
c. Protein misfolding
d. Type 1 TNF receptor

44. Defective apoptosis and increased cell survival is seen in:
a. Autoimmune disease
b. Neurodegenerative diseases
c. Viral infections
d. Ischemic injury

45. Receptor associated kinases 1 (RIP1) and 3 (RIP3) are involved in:
a. Necrosis
b. Apoptosis
c. Necroptosis
d. Pyroptosis

46. Inflammasome is formed in:
a. Necrosis
b. Apoptosis
c. Necroptosis
d. Pyroptosis

47. Fibrinoid necrosis is seen in all of the following except:
a. Malignant hypertension
b. Aschoff's nodule
c. Polyarteritis nodosa
d. Diabetic glomerulosclerosis

48. Dystrophic calcification is/are found in? (PGI type)
a. Monckeberg's medical sclerosis
b. Papillary carcinoma thyroid
c. Hyperparathyroidism
d. Meningioma
e. Vitamin D intoxication

49. Highest telomerase activity is seen in:
a. Stem cells b. Somatic cells
c. Germ cells d. Benign tumors

50. Organelle where H_2O_2 is produced and destroyed is:
a. Peroxisome b. Lysosome
c. Golgi body d. Ribosome

51. Which of these is not responsible for removal of free radicals?
a. Catalase
b. Superoxide Dismutase
c. NADPH oxidase
d. Glutathione peroxidase

36. a, b, d	37. d	38. a	39. d	40. a	41. d	42. c	43. d	44. a	45. c	46. d
47. d	48. a, b, d	49. c	50. a	51. c						

52. Endometrial adenocarcinoma is caused by
a. Hyperplasia b. Atrophy
c. Both d. None

53. Metaplasia of respiratory tract is due to
a. Vitamin E deficiency
b. Vitamin K deficiency
c. Vitamin A deficiency
d. Vitamin D deficiency

54. In cell death, myelin figures are derived from-
a. Nucleus b. Cell membrane
c. Cytoplasm d. Mitochondria

55. The sign of reversible injury in a case of alcoholic liver disease-
a. Loss of cell membrane
b. Nuclear karyolysis
c. Cytoplasmic vacuole
d. Pyknosis

56. Irreversible cell injury is indicated by-
a. Accumulation of calcium in mitochondria
b. Myelin figures
c. ATP depletion
d. Shifting of ribosomes

57. Type of necrosis occurring in brain-
a. Coagulative b. Liquefactive
c. Fibrinoid d. Caseous

58. Fat necrosis is common in-
a. Omentum
b. Breast
c. Retroperitoneal fat
d. All of the above

59. Example of apoptosis is-
a. Councilman bodies
b. Gamma gandy body
c. Russell bodies
d. None

60. Characteristic feature of apoptosis-
a. Cell membrane intact
b. Cytoplasmic eosinophilia
c. Nuclear moulding
d. Cell swelling

61. A 55-years-old male who has a long history of excessive drinking presents with signs of alcoholic hepatitis. Microscopic Examination of a biopsy of this patient's liver reveals irregular eosinophilic hyaline inclusions within the cytoplasm of the hepatocytes. These eosinophilic inclusions are composed of:
a. Immunoglobulin
b. Excess plasma proteins
c. Keratin intermediate filaments
d. Lipofuscin

62. Characteristic feature of apoptosis-(PGI)
a. Cell membrane intact
b. Cytoplasmic eosinophilia
c. Nuclear moulding
d. Cell Shrinkage

63. Lipofuscin granules are examples of
a. Apoptotic bodies
b. Residual bodies
c. Psammomatous calcification
d. Hyaline change

64. Squamous metaplasia is seen in all of the following except
a. Vitamin A deficiency
b. Smoking
c. Gastroesophageal reflux disease
d. Stone obstructing a duct

65. All of the following are true about apoptosis except
a. Cell shrinkage
b. Altered orientation of lipids
c. Only pathological
d. No adjacent inflammation

66. All are true about reversible cell injury except
a. Cellular swelling is the earliest manifestation
b. Organ shows increased turgor and has decreased weight
c. Clear vacuoles represent distended and pinched off ER
d. Increased eosinophilia of cytoplasm

52. c	53. c	54. b	55. c	56. a	57. b	58. d	59. a	60. a	61. c	62. a, d
63. b	64. c	65. c	66. b							

67. Which of the following is/are true about the microscopic appearance of necrotic cells?

a. Increased eosinophilia due to loss of cytoplasmic RNA
b. Increased eosinophilia due to denaturation of cytoplasmic proteins
c. Glassy homogenous appearance due to loss of glycogen particles
d. All of the above

68. Which phenomenon/phenomena characterise irreversibility of cell injury?

a. Mitochondrial dysfunction
b. Profound membrane damage
c. Nuclear disaggregation
d. Both A and B

69. All of the following are pro apoptotic except

a. Smac
b. Diablo
c. BAX
d. BCL- XL

70. All of the following is/are true about extrinsic pathway of apoptosis except

a. Engagement of plasma membrane death receptors
b. Type I TNF receptor is prototype
c. Caspase 9 is classically involved
d. Mechanism of apoptosis of virus infected cells

71. Programmed necrosis is another name for

a. Necroptosis
b. Pyroptosis
c. Atrophy
d. Autophagy

72. Macrophage-specific deletion of which autophagy related gene increases susceptibility to tuberculosis

a. Atg 3
b. Atg 4
c. Atg 5
d. Atg 6

73. Which of the following is false about lipofuscin?

a. Injurious to cell
b. Perinuclear in location
c. Lipid peroxidation
d. Yellow brown granular pigment

74. All are examples of metastatic calcification except

a. Milk Alkali syndrome
b. Hyperparathyroidism
c. Renal osteodystrophy
d. Atherosclerosis

75. Metaplasia arises from reprograming of-

a. Stem cells
b. Stellate cells
c. Squamous cells
d. Columnar cells

76. "Caspase-independent" programmed cell death-

a. Necrosis
b. Necroptosis
c. Apoptosis
d. None

77. Immune complexes mediated necrosis is of which type?

a. Coagulative necrosis
b. Liquefactive necrosis
c. Caseous necrosis
d. Fibrinoid necrosis

78. Stain for fat all except:

a. Oil red O
b. Sudan black
c. Sudan III
d. Congo red

79. The enzyme that protects brain from free radical injury:

a. Superoxide dismutase
b. Catalase
c. Glutathione peroxidase
d. Monoamine oxidase

80. Physiological hyperplasia and hypertrophy are seen simultaneously in-

a. Uterus in pregnancy
b. Skeletal muscle in athletes
c. Breast at puberty
d. Thyroid gland during pregnancy

81. All are reversible injury of cell, except-

a. Vacuole
b. Karyorrhexis
c. Fat accumulation
d. Cell wall swelling

67. d	68. d	69. d	70. c	71. a	72. c	73. a	74. d	75. a	76. b	77. d
78. d	79. a	80. a,c	81. b							

82. Fibrinoid necrosis is seen in following except-
a. Pan
b. Diabetes mellitus
c. Malignant hypertension
d. SLE

83. Esoinophilia in necrosed tissue is due to
a. Coagulation of proteins
b. Denaturation of enzymes
c. Lysosomal damage
d. Mitochondrial damage

84. True about apoptosis are all except-
a. Inflammation is present
b. Chromosomal breakage
c. Clumping of chromatin
d. Cell shrinkage

85. CD-95 is a marker of-
a. Death receptor b. MHC complex
c. T helper cells d. NK cells

86. True about apoptosis is-
a. Energy dependent
b. Self-initiated
c. Swelling of cytoplasm
d. Loss of membrane integrity

87. CD-95 has a major role in-
a. Apoptosis
b. Cell necrosis
c. Interferon activation
d. Proteolysis

88. Which of the following organelles plays a pivotal role in apoptosis?
a. Mitochondria
b. Endoplasmic reticulum
c. Nucleus
d. Golgi apparatus

89. Cytosolic cytochrome C plays an important function in-
a. Apoptosis
b. Cell necrosis
c. Electron transport chain
d. Cell division

90. CD-95 is a marker of-
a. Intrinsic pathway of apoptosis
b. Extrinsic pathway of apoptosis
c. Monocyte
d. Leucocyte

91. In apoptosis, cytochrome C acts through-
a. Apaf-1 b. BCL-2
c. FADD d. TNF

92. Which of the following has a direct role in apoptosis-
a. Nitric oxide b. Adenyl cyclase
c. cAMP d. Cytochrome C

93. The following is a antiapoptotic gene-
a. Bax b. Bad
c. BCL-XL d. Bim

94. SMAC/DIABLO
a. Anti-Apoptotic proteins
b. Induces necrosis most often
c. It can act as both apoptotic and antiapoptotic proteins
d. Pro-apoptotic proteins

95. Which of the following is an antiapoptotic gene?
a. C-myc b. P53
c. BCL-2 d. Bax

96. Brown atrophy is due to accumulation of-
a. Melanin b. Hemosiderin
c. Hematin d. Lipofuscin

97. Which of the following pigments are involved in free radical injury?
a. Lipofuscin b. Melanin
c. Bilirubin d. Hematin

98. Dystrophic calcification is seen in-
a. Milk alkali syndrome
b. Atheromatous plaque
c. Hyperparathyroidism
d. Vitamin A intoxication

82. b	83. a	84. a	85. a	86. a	87. a	88. a	89. a	90. b	91. a	92. d
93. c	94. d	95. c	96. d	97. a	98. b					

99. True about psammoma bodies are all except-

a. Seen in meningioma
b. Concentric whorled appearance
c. Contains calcium deposits
d. Seen in teratoma

100. Calcification of soft tissues without any disturbance of calcium metabolism is called-

a. Ionotrophic calcification
b. Monotrophic calcification
c. Dystrophic calcification
d. Calcium induced calcification

101. Metastatic calcification is not seen in-

a. Kidney tubules
b. Fundal glands of stomach
c. Alveoli of lungs
d. Media of Monckeberg degeneration

102. Apoptosis is inhibited by-

a. P53 b. N-myc
c. RAS d. BCL-2

103. Calcification in necrotic tissue is called-

a. Metastatic calcification
b. Dystrophic calcification
c. Calcinosis
d. Tumoral calcinosis

104. Increase in this patient's serum enzyme (e.g. AST/ALT) Levels are seen in hepatitis. Most likely this results from?

a. Autophagy by lysosomes
b. Clumping of nuclear chromatin
c. Defects in the cell membrane
d. Swelling of the mitochondria

105. Which of the following cells is most likely to have the highest telomerase activity?

a. Endothelial cells b. Germ cells
c. Neutrophils d. Erythrocytes

106. Hypertrophy is a type of –

a. Cell injury
b. Cellular adaptation
c. Carcinoma
d. Cell aging

107. Which finding on electron microscopy indicates irreversible cell injury -

a. Dilatation of endoplasmic reticulum
b. Dissociation of ribosomes from rough endoplasmic reticulum
c. Flocculent amorphous densities in the mitochondria
d. Myelin figures

108. Irreversible cell injury is indicated by-

a. Accumulation of calcium in cytosol
b. Myelin figures
c. ATP depletion
d. Shifting of ribosomes

109. In cell death, myelin figures are derived from -

a. Nucleus b. Cell membrane
c. Cytoplasm d. Mitochondria

110. MI is a type of-

a. Coagulative necrosis
b. Liquefactive necrosis
c. Caseous necrosis
d. Fat necrosis

99. d	100. c	101. d	102. d	103. b	104. c	105. b	106. b	107. c,d	108. a
109. b	110. a								

Cell Adaptation, Injury and Death – Explanations

24. C

Metaplasia is a change in phenotype of differentiated cells, often in response to chronic irritation, that makes cells better able to withstand the stress; usually induced by altered differentiation pathway of tissue stem cells; may result in reduced functions or increased propensity for malignant transformation.

Option A- Both epithelial and mesenchymal metaplasia can be seen

Option B- it is a reversible adaptation

Option D- Squamous metaplasia is the most common type

25. B

Cell size is increased by increase in protein synthesis hence genes unexpressed or silent before are now expressed which leads to an increase in DNA content.

Other statements are true.

26 D

Pleomorphism is a feature of dysplasia and anaplasia, so is loss of polarity.

Other options are true.

Refer to the answer above for more details.

27. C

Change in cell type is metaplasia.

Option A- histological hallmark of malignancy

Option B- disordered growth

Option D- increase in number of cells (not change in cells)

28. C

The normal pseudostratified ciliated columnar epithelium of respiratory tract is replaced by stratified squamous epithelium in response to smoking.

Option A- Barret's esophagus (intestinal metaplasia)

29. A

Squamous metaplasia is a common consequence of vitamin A deficiency. (Lung cancer has been recently implicated with this mechanism)

30. C

Option A-Increase in cell size

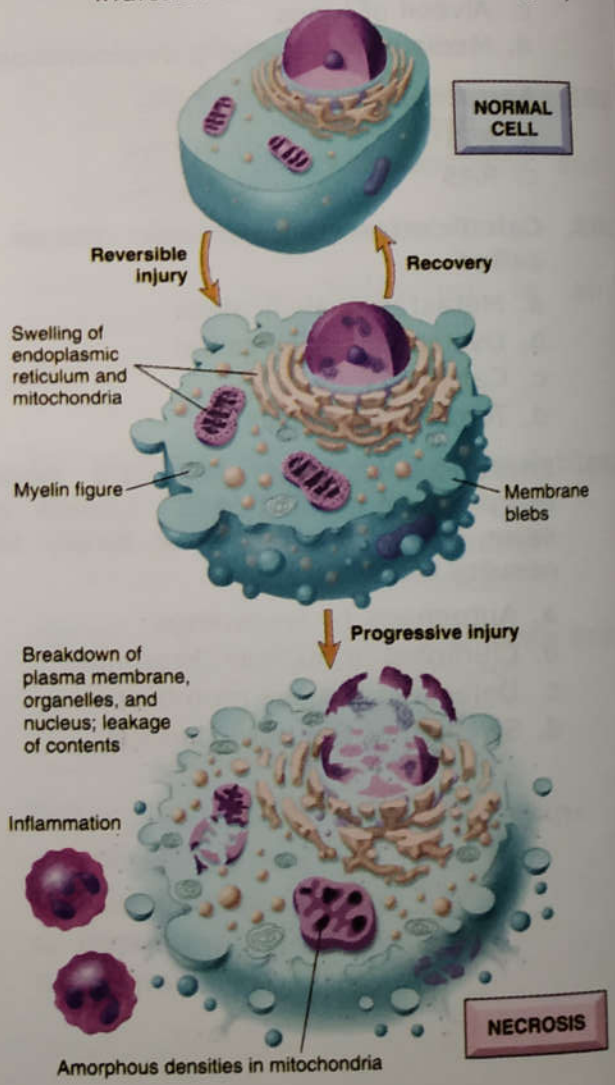
Option B-Increase in cell number

Option C- Cell death

Option D- change in cell type

31. A, D

Reversible to irreversible cell injury is a spectrum and can show similar features. Some however are more indicative of irreversible cell injury.



32. A

Reference : Robbins and Cotran Pathological Basis of Disease, 9th edition

The factors that contribute to reperfusion injury in the myocardium include the following:

- Mitochondrial dysfunction: Mitochondrial permeability increases → apoptosis activated
- Myocyte hypercontracture due to increased levels of intracellular calcium → After reperfusion, the contraction of myofibrils is augmented and uncontrolled, causing cytoskeletal damage and cell death.
- Free radicals, including superoxide anion (•O2 –), hydrogen peroxide (H2O2), hypochlorous acid (HOCl), nitric oxide–derived peroxynitrite, and hydroxyl radicals (•OH)
- Leukocyte aggregation may occlude the microvasculature and contribute to the "no-reflow" phenomenon. Further, leukocytes elaborate proteases and elastases that cause cell death.
- Platelet and complement activation also contribute to microvascular injury. Complement activation is thought to play a role in the no-reflow phenomenon by injuring the endothelium.

33. B

Option A- ATP depletion is one of the earliest biochemical changes occurring in cell injury

Option B- Feature of apoptosis (cell death, not reversible cell injury)

Option C- Cellular swelling and fatty change are earliest findings of reversible cell injury

Option D- Depletion of ATP is a consequence of reduced phosphorylation

34. A

Option A- Although cells of acute inflammation, in some infections they persist even when chronic infection continues like in Pseudomonas

Option B- Seen in parasitic infections and allergic reactions

Option C- Seen in chronic inflammation and viral infections

Option D- Seen in chronic inflammation

35. B

Option .12 A and C are anti apoptotic

Option B- pro apoptotic

Option D- sensor protein

36. A, B, D

Options C and E are not the function of p53 which serves as a guardian of the genome.

37. D

Best option here is option D. Caspases are not the first enzymes activated in apoptosis but they are cysteine proteases involved in protein breakdown leading to apoptosis

38. A

Option A- feature of pyroptosis

Option B- caspase independent programmed cell death

Option C- after death receptor interaction failure to activate caspase 8 can lead to activation of necroptosis

Option D- free radical damage is a part of the mechanism of cell death by necroptosis

39. D

Smac and Diablo are released from the mitochondria when there is increased mitochondrial permeability and they inactivate Inhibitors of Apoptosis (IAPs) which inactivate caspases, thus letting caspases function and promoting apoptosis.

40. A

Caspase 3, 6 and 7 are execution caspases

Caspase 8 is an initiator caspase of extrinsic pathway

Caspase 9 is an initiator caspase of intrinsic pathway

41. D

TUNEL is an acronym for terminal deoxynucleotidyl transferase biotin-dUTP nick end labeling. Terminal deoxynucleotidyl transferase (TdT) is a template-independent DNA polymerase that is normally active in primitive lymphoid cells to increase receptor diversity.

TdT can add random nucleotides to the ends of DNA fragments produced by

endonucleases during apoptosis, since 3′ phosphate groups are the substrate for TdT (3′ phosphate groups are not produced during cell necrosis). Treating tissue sections with TdT and labeled nucleotides then provides a convenient assay for apoptosis. In practice, necrotic debris may nonspecifically trap either fluorescent or chromogenic signals and result in a false-positive assay. Apoptosis can also be detected by means of assays for activated caspase-3, Fas ligand, and annexin V.

42. C

Option C- Pro apoptotic

Options A, B, D- Sensor of apoptosis

43. D

Option A, B, C- intrinsic pathway

Option D- death receptor or extrinsic pathway

44. A

Defective apoptosis and increased cell survival of cells which should have died is seen in autoimmune disease and cancer.

Increased apoptosis and decreased cell survival of cells which should not have died is seen in the other three options.

45. C

Interaction of TNFR and inactivation of caspase 8 leads to activation of RIP1 and RIP3 to cause necroptosis

Option A- lysosomal enzymes

Option C- Caspases 3, 6, 8, 9, 10

Option D- Caspases 1 and 11

46. D

Microbial products that enter the cytoplasm of infected cells are recognized by cytoplasmic innate immune receptors and can activate the multiprotein complex called the inflammasome. The function of the inflammasome is to activate caspase1, (also known as interleukin-1β converting enzyme) which cleaves a precursor form of IL-1 and releases its biologically active form. IL-1 is a mediator of many aspects of inflammation, including leukocyte recruitment and fever. Caspase-1 and, more importantly, the closely related caspase-11 also induce death of the cells. Unlike classical apoptosis, this pathway of cell death is characterized by swelling of cells, loss of plasma membrane integrity, and release of inflammatory mediators. Pyroptosis results in the death of some microbes that gain access to the cytosol and promotes the release of inflammasome-generated IL-1.

47. D

Fibrinoid necrosis is a special form of necrosis usually seen in immune reactions involving blood vessels. This pattern of necrosis typically occurs when complexes of antigens and antibodies are deposited in the walls of arteries. Deposits of these "immune complexes," together with fibrin that has leaked out of vessels, result in a bright pink and amorphous appearance in H&E stains, called "fibrinoid" (fibrin-like) by pathologists. This pattern of necrosis is seen classically in options A, B and C as well as rejection of transplants.

48. A, B, D

Dystrophic calcification seen in dying tissues with normal calcium levels.

Option C and E are examples of metastatic calcification seen in normal tissues with increased calcium levels.

49. C

Telomere length is maintained by nucleotide addition mediated by an enzyme called telomerase. Telomerase is a specialized RNA-protein complex that uses its own RNA as a template for adding nucleotides to the ends of chromosomes. Telomerase activity is expressed in germ cells and is present at low levels in stem cells, but it is absent in most somatic tissues

50 A

Properties	O_2	H_2O_2	*Oh
Mechanisms of production	Incomplete reduction of O_2 during oxidative phosphorylation; by phagocyte oxidase in leukocytes	Generated by SOD from O_2 and by oxidases in peroxisomes	Generated from H2O by hydrolysis, e.g., by radiation; from H_2O_2 by Fenton reaction; from O_2

Mechanisms of inactivation	Conversion to H_2O_2 and O_2 by Sod	Conversion to H_2O and O_2 by catalase (peroxisomes), glutathione peroxidase (cytosol, mitochondria)	Conversion to H_2O by glutathione peroxidase
Pathologic effects	Stimulates production of degradative enzymes in leukocytes and other cells; may directly damage lipids, proteins, DNA; acts close to site of production	Can be converted to *OH and OCl_ , which destroy microbes and cells; can act distant from site of production	Most reactive oxygen-derived free radical; principal ROS responsible for damaging lipids, proteins, and DNA

51. C

helps to generate free radicals especially in neutrophils

52. C

Type I endometrial adenocarcinoma arises in a hyperplastic endometrium whereas Type II may arise in an atrophic endometrium

53. C

In the habitual cigarette smoker, the normal ciliated columnar epithelial cells of the trachea and bronchi are often replaced by stratified squamous epithelial cells. Stones in the excretory ducts of the salivary glands, pancreas, or bile ducts, which are normally lined by secretory columnar epithelium, may also lead to squamous metaplasia by stratified squamous epithelium. A deficiency of vitamin A (retinoic acid) induces squamous metaplasia in the respiratory epithelium.

54. B

Phospholipids derived myelin figures are more characteristic for irreversible than reversible cell injury.

55. C

Cellular swelling and organelle swelling are signs of reversible cell injury

56. A

Influx of calcium into mitochondria increases its permeability thus causing a cascade of changes responsible for cellular death.

57. B

Enzymatic digestion by both enzymes released from dying cells and inflammatory cells causes lysis of the dead cells into a liquefied viscous mass- liquefactive necrosis.

Option A- Most common pattern of ischemic necrosis in all organs other than brain

Option C- Usually in vasculitis

Option D- Central necrosis leading to cheese like necrosis on gross

58. D

All sites are fat rich and hence digestion of fatty cells and their membranes followed by saponification into calcium salts is relatively common in these sites.

59. A

Option B- Calcified bodies seen in congestive splenomegaly

Option C- Intracellular accumulation of Igs in plasma cells

60. A

Intact cell membrane and no inflammation is characteristic of apoptosis. Other features are more classical for necrosis.

61. C

CK 8/18- Mallory hyaline

62. A, D

Option B- feature of necrosis more than apoptosis

63. B

Autophagy ("self-eating") is the process in which the starved cell eats its own components in an attempt to reduce nutrient demand to match the

supply. Some of the cell debris within the autophagic vacuoles may resist digestion and persist in the cytoplasm as membrane-bound residual bodies. An example of residual bodies is lipofuscin granules.

64. C

Options A, B, D are examples of change of columnar epithelium to squamous. However GERD in esophagus causes change of squamous to columnar (called Intestinal Metaplasia or Barret's Esophagus)

65. C

Feature	Necrosis	Apoptosis
Cell size	Enlarged (swelling)	Reduced (shrinkage)
Nucleus	Pyknosis → karyorrhexis → karyolysis	Fragmentation into nucleosome size fragments & condensation of chromatin
Plasma membrane	Disrupted	Intact; altered structure, especially orientation of lipids
Cellular contents	Enzymatic digestion; may leak out of cell	Intact; may be released in apoptotic bodies
Adjacent inflammation	Frequent	No
Physiologic or pathologic role	Invariably pathologic (culmination of irreversible cell injury)	Often physiologic, means of eliminating unwanted cells; may be pathologic after some forms of cell injury, especially DNA damage

66. B

- Cellular swelling is the first manifestation of almost all forms of injury to cells.
- It is a difficult morphologic change to appreciate with the light microscope; it may be more apparent at the level of the whole organ.
- When it affects many cells, it causes some pallor, increased turgor, and increase in weight of the organ.
- On microscopic examination, small clear vacuoles may be seen within the cytoplasm; these represent distended and pinched-off segments of the ER → Hydropic change or vacuolar degeneration.
- Swelling of cells is reversible.
- Cells may also show increased eosinophilic staining, which becomes much more pronounced with progression to necrosis

67. D

Necrotic cells show increased eosinophilia in hematoxylin and eosin (H & E) stains, attributable in part to the loss of cytoplasmic RNA (which binds the blue dye, hematoxylin) and in part to denatured cytoplasmic proteins (which bind the red dye, eosin). The necrotic cell may have a more glassy homogeneous appearance than do normal cells, mainly as a result of the loss of glycogen particles. When enzymes have digested the cytoplasmic organelles, the cytoplasm becomes vacuolated and appears moth-eaten.

68. D

Two phenomena consistently characterize irreversibility—the inability to reverse mitochondrial dysfunction (lack of oxidative phosphorylation and ATP generation) even after resolution of the original injury, and profound disturbances in membrane function. Injury to lysosomal membranes results in the enzymatic dissolution of the injured cell that is characteristic of necrosis.

69. D

- Smac/Diablo, enter the cytoplasm, where they bind to and neutralize

cytoplasmic proteins that function as physiologic inhibitors of apoptosis (called IAPs).

- The normal function of the IAPs is to block the activation of caspases, including executioners like caspase-3, and keep cells alive. Thus, the neutralization of these IAPs permits the initiation of a caspase cascade.
- BAX and BAK are the two prototypic members of the BCL2 family.
- BCL XL, MCL1 and BCL2 are the anti apoptotic members

70. C

Caspase 8 and 10 (in humans) are involved in the extrinsic pathway

71. A

The following features characterize necroptosis:

- Morphologically, and to some extent biochemically, it resembles necrosis, both characterized by loss of ATP, swelling of the cell and organelles, generation of ROS, release of lysosomal enzymes and ultimately rupture of the plasma membrane as discussed earlier.
- Mechanistically, it is triggered by genetically programmed signal transduction events that culminate in cell death. In this respect it resembles programmed cell death, which is considered the hallmark of apoptosis

Because of the duality of these features, necroptosis is sometimes called programmed necrosis to distinguish it from the more usual forms of necrosis driven passively by toxic or anoxic injury to the cell.

In sharp contrast to apoptosis, the genetic program that drives necroptosis does not result in caspase activation and hence it is also sometimes referred to as "caspase-independent" programmed cell death.

72. C

Many pathogens are degraded by autophagy; these include mycobacteria, Shigella spp., and HSV-1. This is one way by which microbial proteins are digested and delivered to antigen presentation pathways. Macrophage-specific deletion of Atg5 increases susceptibility to tuberculosis.

73. A

Lipofuscin is not injurious to the cell or its functions. Its importance lies in its being a telltale sign of free radical injury and lipid peroxidation. The term is derived from the Latin (fuscus, brown), referring to brown lipid. In tissue sections it appears as a yellow-brown, finely granular cytoplasmic, often perinuclear, pigment.

74. D

There are four principal causes of hypercalcemia:

(1) increased secretion of parathyroid hormone (PTH) with subsequent bone resorption, as in hyperparathyroidism due to parathyroid tumors, and ectopic secretion of PTH-related protein by malignant tumors;

(2) resorption of bone tissue, secondary to primary tumors of bone marrow (e.g., multiple myeloma, leukemia) or diffuse skeletal metastasis (e.g., breast cancer), accelerated bone turnover (e.g., Paget disease), or immobilization;

(3) vitamin D–related disorders, including vitamin D intoxication, sarcoidosis (in which macrophages activate a vitamin D precursor), and idiopathic hypercalcemia of infancy (Williams syndrome), characterized by abnormal sensitivity to vitamin D; and

(4) renal failure, which causes retention of phosphate, leading to secondary hyperparathyroidism.

Less common causes include aluminum intoxication, which occurs in patients on chronic renal dialysis, and milk-alkali syndrome, which is due to excessive ingestion of calcium and absorbable antacids such as milk or calcium carbonate

75. A

Refer to answer above for details.

76. B

Refer to explanation above

77. D

Refer to explanation above

78. D
Option D is a stain used for Amyloid

79. A (single best answer)

80. A, C

81. B
Refer to the explanation provided before

82. B
Refer to the explanation provided above

83 A
Refer to the explanation provided above

84. A
Refer to the explanation provided above

85. A
Refer to the explanation provided above

86. A

87 A
Refer to the explanation provided above

88. A
Refer to the explanation provided above

89. A
Refer to the explanation provided above

90. B
Refer to the explanation provided above

91 A
Refer to the explanation provided above

92 D

93. C

94. D

95. C

96. D

97. A

98. B

99. D

100. C

101. D

102. D

103. B

104 C

105. B

106. B

107. C, D

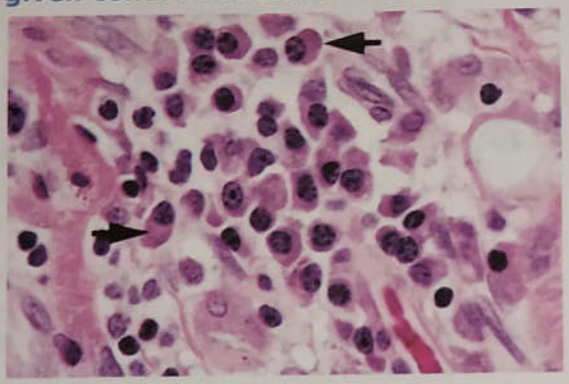
108. A (single best option)

109. B

110. A

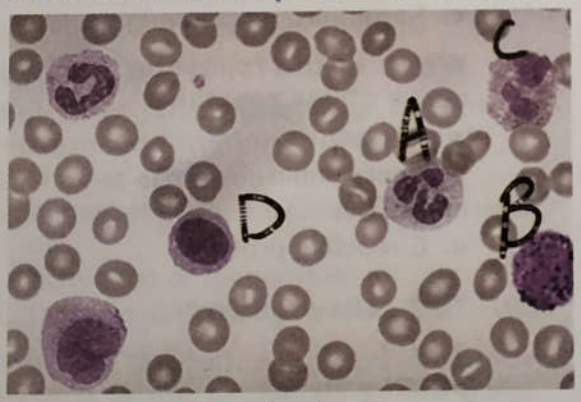
Inflammation, Repair and Hemodynamics – Questions

111. Identify the arrow marked cell in the given condition below?



a. Macrophage
b. Lymphocyte
c. Plasma cell
d. Eosinophil

112. Which of the following cells will increase in case of parasite infection?



a. A
b. B
c. C
d. D

113. Vasodilation in acute inflammation is seen earliest in:

a. Venules
b. Arterioles
c. Capillaries
d. Vein

114. Increased permeability in acute inflammation is due to:

a. Histamine
b. IL-2
c. TGFβ
d. FGF

115. Sequence of events in acute inflammation:

a. Vasodilatation → Stasis → Transient vasoconstriction → Increased permeability
b. Transient vasoconstriction → Increased permeability → Stasis → Vasodilatation
c. Transient vasoconstriction → Vasodilatation → Stasis → Increased permeability
d. Transient vasoconstriction → Vasodilatation → Increased permeability → Stasis

116. The following is not an adhesion molecule?

a. Spectrin
b. Integrins
c. Selectins
d. Cadherin

117. The RBCs with schizonts of P. Falciparum are not visible on peripheral blood smear due to which of the following reason?

a. Capillary adherence or sequestration of parasitized RBCs
b. ADCC mediated RBC destruction
c. Selective hemolysis of affected RBCs in spleen
d. Cellular lysis due to hemozoin produced by the parasites Erythrocyte changes in Malaria

118. Amino acid which is useful in Neutrophil extracellular traps (NETs) as to causes lysis of chromatin is?

a. Arginine
b. Alanine
c. Phenyl alanine
d. Valine

111. d	112. c	113. b	114. a	115. d	116. a	117. a	118. a

119. The following statements are true regarding neutrophil extracellular trap:

a. Produced by neutrophils in response to infectious pathogens and inflammatory mediators
b. Provide a high concentration of antimicrobial substances at sites of infection
c. Prevent the spread of the microbes by trapping them in the fibrils
d. All of the above

120. All of the following vascular changes are observed in acute inflammation, except:

a. Vasodilation
b. Stasis of blood
c. Increased vascular permeability
d. Decreased hydrostatic pressure

121. Correct sequence in extravasation of leukocytes is:

a. Margination – rolling – adhesion – transmigration
b. Transmigration – margination – rolling – adhesion
c. Rolling – adhesion – transmigration – margination
d. Adhesion – transmigration – margination – rolling

122. Transmigration of WBC across the endothelium is called:

a. Margination
b. Rolling
c. Pavementing
d. Diapedesis

123. Which of the following is an alpha chemokine?

a. IL-8
b. MCP-1
c. Eotaxin
d. Lymphotactin

124. Which selectin is stored in weibel palade bodies?

a. Cadherins
b. P-Selectin
c. E-Selectins
d. L-Selectin

125. Which of the following enzymes are responsible for generating 'oxygen burst' within neutrophils for killing intracellular bacteria?

a. Superoxide dismutase
b. Glutathione peroxidase
c. Oxidase
d. Catalase

126. Which of the following is an Opsonin?

a. C3a
b. C3b
c. C5a
d. LTC4

127. Which of the cell derived chemical mediators of inflammation is/ are preformed in secretory granules?(PGI)

a. Histamine
b. Lysosomal enzymes
c. Leukotrienes
d. Prostaglandins
e. Serotonin

128. Which of the following is a negative acute phase reactant?

a. Albumin
b. Haptaglobin
c. Ferritin
d. C reactive protein

129. Th 1 cells produce?

a. IL-1
b. IFN gamma
c. IL-4
d. IL-5

130. Primary granules of neutrophil have?

a. Proteinase 3
b. Alkaline phosphatase
c. Acid protease
d. Lactoferrin

131. Major basic protein is found in?

a. Macrophage
b. Eosinophils
c. Basophil
d. Neutrophil

119. d	120. d	121. a	122. d	123. a	124. b	125. c	126. b	127. a,b,e
128. a	129. b	130. a	131. b					

132. The following are labile cells except:
a. Hepatocytes
b. Bone marrow
c. Intestinal mucosa
d. Epithelium of skin

133 Liver biopsy in chronic RHF shows?
a. Shrinking
b. Greasy fatty liver
c. Hypertrophy
d. Congestion

134 Heart failure cells are:
a. Foam cells
b. Lipid laden macrophages
c. Hemosiderin laden macrophages
d. Type 1 pneumocytes

135 Hypercoagulability due to defective factor V gene is called:
a. Lisbon mutation
b. Leiden mutation
c. Antiphospholipid syndrome
d. Inducible thrombocytopenia syndrome

136 Which of the following is not a recognised function of thrombin?
a. Platelet activation
b. Adhesion of neutrophils to endothelium
c. Activation of monocytes
d. Inactivation of lymphocytes

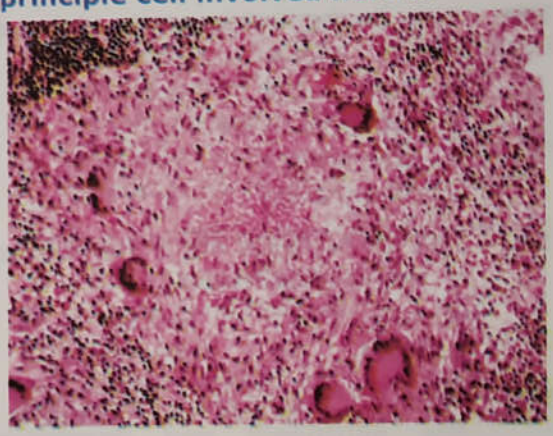
137 Which of the following glycosaminoglycans is not produced in the golgi apparatus?
a. Heparan sulphate
b. Dermatan sulphate
c. Keratan sulphate
d. Hyaluronan

138 Healing tissue has which type of collagen
a. I
b. II
c. III
d. IV

139 In mediating acute phase response of inflammation which of the following does not play a major role?
a. IL-1
b. IL-6
c. IL-13
d. TNF

140 Which of the following is not a major pyrogen in inflammation?
a. IL-1
b. PGE2
c. TNF
d. IFN

141 The following histopathological picture is from a patient with cough, night sweats and a cheesy white focus in left upper lobe of lung. The principle cell involved here is



a. Lymphocyte
b. Histiocyte
c. Eosinophil
d. Multinucleated giant cell

142 Eosinophillia is found in?
a. Cryptococcus
b. HPV
c. Stronglyloides
d. Typhoid

143 Leukocyte migration through endothelium is induced by:
a. Selectin
b. N CAM
c. CAM
d. PECAM

144 Role of P-selectin in inflammation-
a. Rolling
b. Adhesion
c. Homing
d. Transmigration

145 What decreases during acute inflammation?
a. Ceruloplasmin
b. Transferrin
c. CRP
d. ESR
e. Albumin

132. a	133. d	134. c	135. b	136. d	137. d	138. c	139. c
140. d	141. b	142. c	143. d	144. a	145. e		

146 Which of the following is the chief mediator associated with resetting the hypothalamic temperature set point at a higher level, resulting in fever?

a. PGF2 alpha b. PGE1
c. PGE2 d. PGI2

147 Which of the following is a major pyrogenic cytokine: (PGI)

a. IL-12 b. TNF
c. IFN-Y d. Bradykin

148 Pain during inflammation is mediated by:

a. Nitric oxide
b. Leukotriene B4
c. Bradykinin
d. Chemokines

149 The following is not a chemoattactant in acute inflammation:

a. IL-8 b. C5a
c. LTB4 d. Kinins

150 Most important cytokine for the synthesis and deposition of connective tissue proteins:

a. TGF-α b. TGF-β
c. FGF-1 d. FGF-2

151 TNF and IL1 are produced by:

a. Neutrophils
b. Monocytes
c. Lymphocytes
d. Activated Macrophages

152 Stellate Granuloma are seen in:

a. Cat scratch disease
b. Sarcoidosis
c. LGV
d. Histoplasmosis

153 A 9 years old boy is admitted with acute abdominal pain localized in the right illac fossa. He is pyrexia with localized peritonism in RIF. The causative cell involved here:

a. Lymphocytes
b. Neutrophil
c. Macrophages
d. Monocytes

154 True statement about alternative macrophage activation (PGI type)

a. Induced by the cytokine IFN-y
b. Kill ingested organisms
c. Secrete cytokines that stimulate inflammation
d. Main function is in tissue repair
e. Anti inflammatory

155 The principle cell in granuloma

a. Fibroblast
b. Epithelioid cell
c. Giant cell
d. Plasma cell

156 Durck granuloma is seen in:

a. Congenital syphilis
b. Cat scratch disease
c. Histoplasmosis
d. Cerebral malaria

157 Hallmark of chronic inflammation:

a. Increased vascular permeability
b. Vasodilation
c. Granulomas
d. Tissue destruction

158 Granulomatous inflammation seen in A/E:

a. Syphilis
b. Tuberculosis
c. Sarcoidosis
d. AIDS

159 Necrotizing epitheloid cell granulomas are seen in all, except:

a. Tuberculosis
b. Wegener's granulomatosis
c. Cat's Scratch disease
d. Leprosy

160 Cell involved in the immediate phase of wound healing:

a. Platelets
b. Fibroblasts
c. Basophils
d. Macrophages

146. c	147. b, c	148. c	149. d	150. b	151. d	152. a	153. b	154. a,d,e
155. b	156. d	157. d	158. d	159. d	160. a			

161 Scar contraction is caused by?

a. Fibroblasts
b. Myofibroblasts
c. Epithelial cells
d. None

162 Find the false statement regarding wound healing:

a. Macrophages are the key cellular constituents
b. TGF-β is the most important fibrogenic agent
c. In remodelling type III collagen replaces type I collagen
d. Vitamin C is required for the hydroxylation of procollagen

163 In a sutured incised wound, re-epithelialization is complete by:

a. 24 hours
b. 48 hours
c. 5 days
d. 2 weeks

164 Permanent tissue is?

a. Heart
b. Liver
c. Kidney
d. Skeletal muscle

165 Which of the following DOES NOT present with recurrent episodes of upper limb thrombosis:

a. Prostatic Cancer
b. Pancreatic Cancer
c. Osteosarcoma
d. Acute promyeloytic leukemia

166 Endothelium activation refers to:

a. Aberration of anatomy of vessel wall
b. Irreversible changes in functional state of vessel wall
c. Smooth muscle proliferation
d. Increased expression of adhesion molecules for leukocyte recruitment

167 Weibel-palade bodies are present in:

a. Vascular endothelial cells
b. Warthin finkeldey cells
c. Leydig cells
d. Dendritic cells

161. b	162. c	163. c	164. a	165. c	166. d	167. a

Inflammation, Repair and Hemodynamics – Explanations

111.D

Plasma cell is a mononuclear cell with eccentric nucleus and a perinuclear halo.

Option A- mononuclear cell with abundant cytoplasm and pale nucleus

Option B- mononuclear cell with scanty cytoplasm and dense nucleus

Option D- bilobed nucleus with eosinophilic coarse granules

112.C

Option A- Neutrophil- increased in bacterial infections

Option B- Basophil- increased in myeloproliferative neoplasms

Option D- Lymphocyte- increased in viral infections

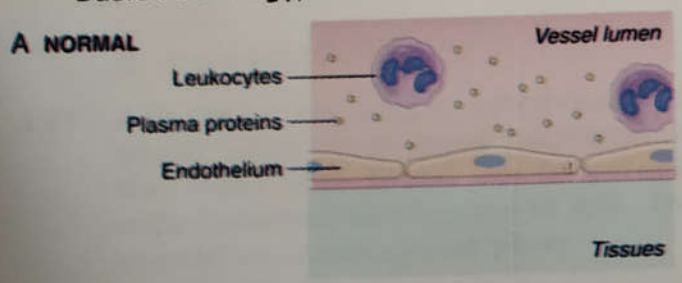
113.B

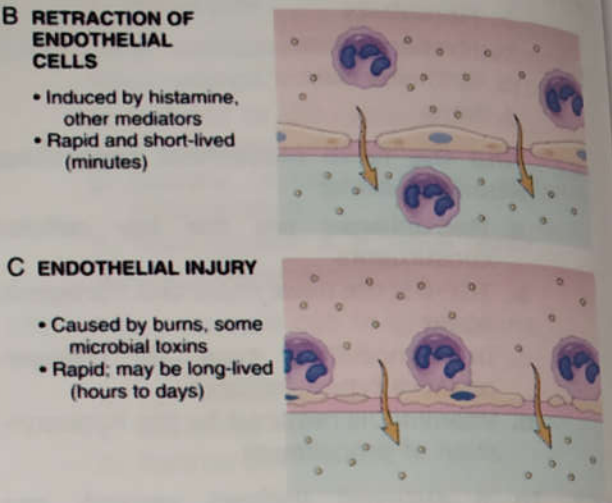
Reference: Robbins and Cotran Pathological Basis of Disease, 9th edition and Robbins Basic Pathology, 10th edition

Vasodilation first involves the arterioles and then leads to opening of new capillary beds in the area. The result is increased blood flow, which is the cause of heat and redness (erythema) at the site of inflammation

114.A

Reference: Robbins and Cotran Pathological Basis of Disease, 9th edition and Robbins Basic Pathology, 10th edition




B RETRACTION OF ENDOTHELIAL CELLS
• Induced by histamine, other mediators
• Rapid and short-lived (minutes)
C ENDOTHELIAL INJURY
• Caused by burns, some microbial toxins
• Rapid; may be long-lived (hours to days)

115 D

Vasodilation (earliest in arterioles) → Increased permeability→ Exudative fluid leaks out → Edema → Slower blood flow → Hemoconcentration and Increased viscosity → Stasis → Margination

116 A

Spectrin is a part of the RBC cytoskeletal whereas the other options are cellular adhesion molecules involved in various processes of cell growth, differentiation and inflammation.

117 A

Reference: Robbins and Cotran Pathological Basis of Disease, 9th edition and Robbins Basic Pathology, 10th edition

P. falciparum causes infected red cells to clump together (rosette) and to stick to endothelial cells lining small blood vessels (sequestration), which blocks blood flow. Several proteins, including P. falciparum erythrocyte membrane protein 1 (PfEMP1), associate and form knobs on the surface of red cells. PfEMP1 binds to ligands on endothelial cells, including CD36, thrombospondin, VCAM-1, ICAM-1, and E-selectin. Red cell sequestration

decreases tissue perfusion and leads to ischemia, which is responsible for the manifestations of cerebral malaria, the major cause of death in children with malaria

118 A

The process is thought to begin with NADPH oxidase activation of protein-arginine deiminase 4 (PAD4) via reactive-oxygen species (ROS) intermediaries. PAD4 is responsible for the citrullination of histones in the neutrophil, resulting in decondensation of chromatin.

119.D

Neutrophil extracellular traps (NETs) are extracellular fibrillar networks that concentrate anti-microbial substances at sites of infection and prevent the spread of the microbes by trapping them in the fibrils.

They are produced by neutrophils in response to infectious pathogens (mainly bacteria and fungi) and inflammatory mediators (e.g., chemokines, cytokines, and complement proteins).

The extracellular traps consist of a viscous meshwork of nuclear chromatin that binds and concentrates granule proteins such as anti-microbial peptides and enzymes.

NETs provide an additional mechanism of killing microbes that does not involve phagocytosis.

In the process of NET formation, the nuclei of the neutrophils are lost, leading to the death of the cells, sometimes called NETosis, representing a distinctive form of cell death affecting neutrophils.

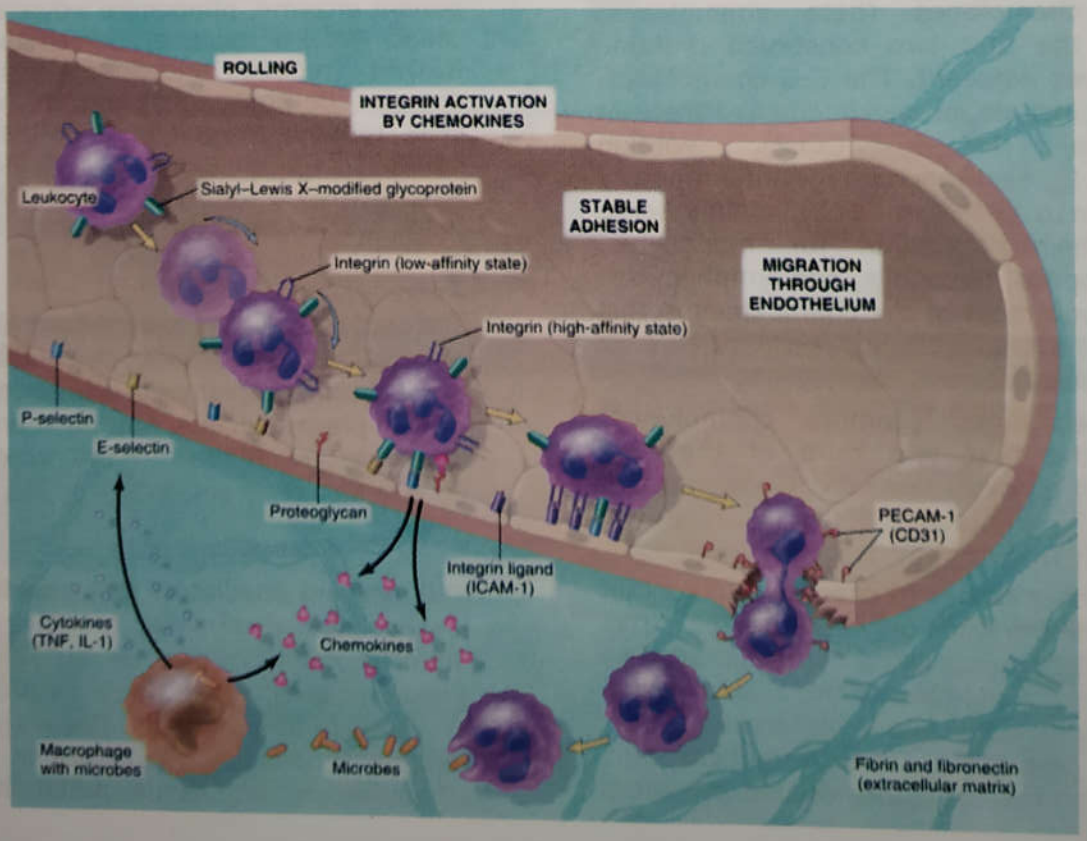
NETs also have been detected in the blood during sepsis.

The nuclear chromatin in the NETs, which includes histones and associated DNA, may be a source of nuclear antigens in systemic autoimmune diseases, particularly lupus, in which individuals react against their own DNA and nucleoproteins

120.D

Refer to the answer above (decreased oncotic pressure is seen leading to edema, not decreased hydrostatic pressure)

121.A



122. D

Option A and C- arrangement of leucocytes towards the margins of the vessel following increased permeability and loss of fluid

Option B- unstable tethering of the leucocyte over the endothelial surface

123 A

Chemokines are a family of small (8–10 kD) proteins that act primarily as chemoattractants for specific types of leukocytes. About 40 different chemokines and 20 different receptors for chemokines have been identified. They are classified into four major groups

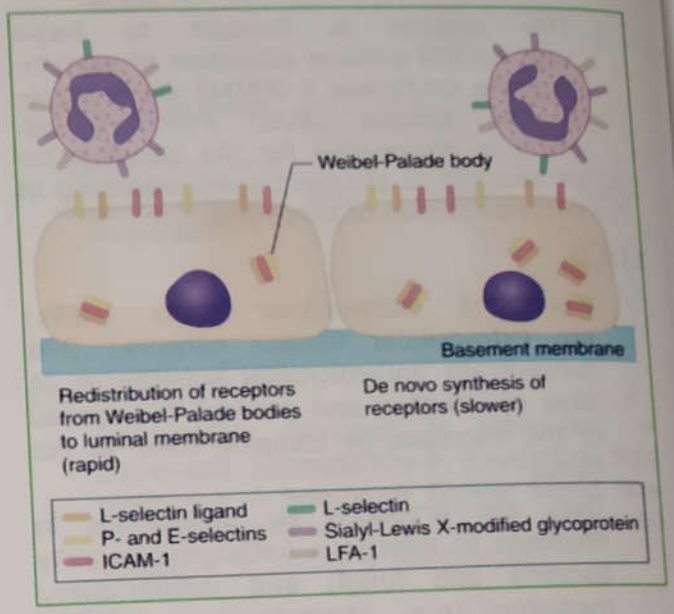
C-X-C chemokines (alpha chemokines) have one amino acid residue separating the first two of the four conserved cysteines. These chemokines act primarily on neutrophils. IL-8 (now called CXCL8) is typical of this group. It is secreted by activated macrophages, endothelial cells, and other cell types, and causes activation and chemotaxis of neutrophils, with limited activity on monocytes and eosinophils. Its most important inducers are microbial products and cytokines, mainly IL-1 and TNF.

C-C chemokines (beta chemokines) have the first two conserved cysteine residues adjacent. The C-C chemokines, which include monocyte chemoattractant protein (MCP-1, CCL2), eotaxin (CCL11), and macrophage inflammatory protein1α (MIP-1α, CCL3), mainly serve as chemoattractants for monocytes, eosinophils, basophils, and lymphocytes. Although most of the chemokines in this class have overlapping actions, eotaxin selectively recruits eosinophils.

C chemokines (gamma chemokines) lack the first and third of the four conserved cysteines. The C chemokines (e.g., lymphotactin, XCL1) are relatively specific for lymphocytes.

CX3C chemokines (d chemokines) contain three amino acids between the CL1). This chemokine exists infirst two cysteines. The only known member of this class is called fractalkine (CX3) two forms: a cell surface-bound protein induced on endothelial cells by inflammatory cytokines that promotes strong adhesion of monocytes and T cells, and a soluble form, derived by proteolysis of the membranebound protein, that has potent chemoattractant activity for the same cells

124. B



125. C

ROS are produced by the rapid assembly and activation of a multicomponent enzyme, phagocyte oxidase (also called NADPH oxidase), which oxidizes NADPH (reduced nicotinamide-adenine dinucleotide phosphate) and, in the process, reduces oxygen to the superoxide anion ($O_2^{\bullet}$). In neutrophils,this oxidative reaction is tightly linked to phagocytosis, and is called the respiratory burst.

126. B

The major opsonins are immunoglobulin (Ig)G antibodies, the C3b breakdown product of complement activation, and certain plasma lectins, notably mannose-binding lectin, all of which are recognized by specific receptors on leukocytes

127. A, B, E

Cell derived mediators are rapidly released from intracellular granules (e.g., amines) or are synthesized de novo (e.g., prostaglandins, leukotrienes, cytokines) in response to a stimulus. The major cell types that produce mediators of acute inflammation are tissue macrophages,

dendritic cells, and mast cells, but platelets, neutrophils, endothelial cells, and most epithelia also can be induced to elaborate some of the mediators. Therefore, cell-derived mediators are most important for reactions against offending agents in tissues. Plasma-derived mediators (e.g., complement proteins) are present in the circulation as inactive precursors that must be activated, usually by a series of proteolytic cleavages, to acquire their biologic properties. They are produced mainly in the liver, are effective against circulating microbes, and also can be recruited into tissues.

128. A

Negative acute-phase proteins

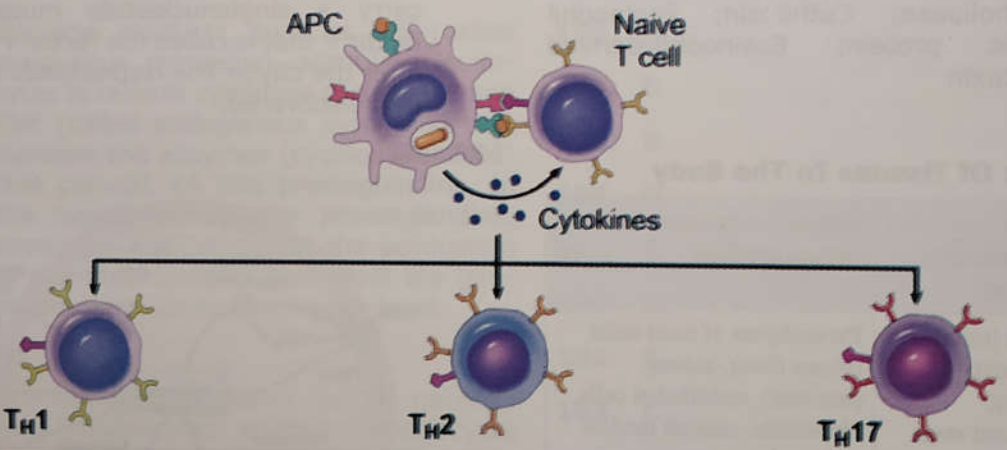
The liver responds by producing a large number of APRs. At the same time, the production of a number of other proteins is reduced; these are therefore referred to as "negative" APPs. Negative APPs are albumin, transferrin, transthyretin, transcortin, and retinol-binding protein.

Positive acute-phase proteins

Positive APPs are CRP, D-dimer protein, mannose-binding protein, alpha 1 antitrpysin, alpha 1 antichymotrypsin, alpha 2 macroglobulin, fibrinogen, prothrombin, factor VIII, von-Willebrand factor, plasminogen, complement factors, ferritin, SAP complement, SAA, ceruloplasmin (Cp), and haptoglobin (Hp).

Positive APPs serve different physiological functions for the immune system. Some act to destroy or inhibit growth of microbes, e.g., CAA and Hp. Others give negative feedback on the inflammatory response, e.g., serpins, alpha 2 macroglobulin and coagulation factors affect coagulation. Positive APPs are produced during the APR associated with anorexia and changed metabolism.

129. B



Major cytokines produced	IFN-γ	IL-4, IL-5, IL-13	IL-17, IL-22
Cytokines that induce this subset	IFN-γ, IL-12	IL-4	TGF-β, IL-6, IL-1, IL-23
Immunological reactions triggered	Macrophage activation, stimulation of lgG antibody production	Stimulation of lgE production, activation of mast cells and eosinophils	Recruitment of neutrophils, monocytes
Host defense against	Intracellular microbes	Helminthic parasites	Extracellular bacteria, fungi
Role in disease	Autoimmune and other chronic inflammatory diseases (such as IBD, psoriasis, granulomatous inflammation)	Allergies	Autoimmune and other chronic inflammatory diseases (such as IBD, psoriasis, MS)

130. A

Azurophilic (primary)granules (formed in promyelocyte stage)

Lysosomal enzymes: acid hydrolases, acid phosphatase, β-glucuronidase, proteinase 3

Myeloperoxidase

Elastase

Arylsulfatase

Cationic antibacterial proteins

Specific (secondary) granules (formed in myelocyte stage)

Lysozyme

Lactoferrin

Collagenase

Plasminogen activator

Aminopeptidase

131. B

Larger granules of eosinophils contain- Major basic protein; Ac d hydrolases; Peroxidase;

Phospholipase; Cathe sin; Eosinophil cationic protein; Eosinophil-derived neurotoxin

132. A

Types Of Tissues In The Body

Labile (continuously dividing)	Hematopoietic cells and surface epithelia
Stable (minimal proliferative activity, quiescent stage G0)	Parenchyma of most solid tissues (liver, kidney, pancreas), endothelial cells, fibroblasts, smooth muscle cells
Permanent tissues (terminally differentiated; non proliferative)	Majority of neurons and cardiac muscle cells (skeletal muscle cells too- but harbor adult tissue stem cells as well)

133. D

Congestion of the hepatic and portal vessels may produce pathologic changes in the liver, the spleen, and the GI tract. The liver is usually increased in size and weight (congestive hepatomegaly) due to prominent passive congestion, greatest around the central veins. Grossly, this is reflected as congested red-brown pericentral zones, with relatively normal-colored tan periportal regions, producing the characteristic "nutmeg liver" appearance

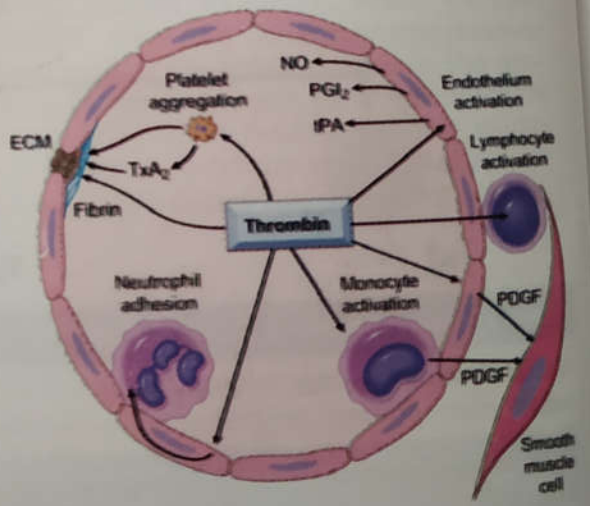
134. C

Congested tissues take on a dusky reddish-blue color (cyanosis) due to red cell stasis and the presence of deoxygenated hemoglobin. Microscopically, acute pulmonary congestion exhibits engorged alveolar capillaries, alveolar septal edema, and focal intraalveolar hemorrhage. In chronic pulmonary con gestion, which is often caused by congestive heart failure, the septa are thickened and fibrotic, and the alve oli often contain numerous hemosiderin-laden macrophages called heart failure cells

135. B

Approximately 2% to 15% of Caucasians carry a singlenucleotide mutation in factor V that is called the factor V Leiden, after the city in The Netherlands where it was discovered.

136. D



137. D

4 distinct families of GAGs

Heparan Sulphate

Chondroitin/Dermatan Sulphate

Keratan Sulphate

Hyaluronan (HA)

First three synthesized and assembled in the Golgi apparatus and RER as proteoglycans

HA is produced at the plasma membrane by enzymes and is not linked to a protein backbone

138. C

Option A- scar tissue

Option D- basement membrane

139. C

Acute-phase proteins are plasma proteins, mostly synthesized in the liver, whose plasma concentrations may increase several hundredfold as part of the response to inflammatory stimuli. Three of the bestknown of these proteins are Creactive protein (CRP), fibrinogen, and serum amyloid A (SAA) protein. Synthesis of these molecules in hepatocytes is stimulated by cytokines, especially IL6 (for CRP and fibrinogen) and IL1 or TNF (for SAA).

140. D

Bacterial products, such as LPS (called exogenous pyrogens), stimulate leukocytes to release cytokines such as IL1 and TNF (called endogenous pyrogens) that increase the enzymes (cyclooxygenases) that convert AA into prostaglandins. In the hypothalamus, the prostaglandins, especially PGE2stimulate the production of neurotransmitters that reset the temperature set point at a higher level.

141. B

The photograph shows central necrosis with lymphocytes, epithelioid histiocytes and Langhan's type of giant cell. Principle cell of a granuloma is an epithelioid histiocyte formed by activation of macrophages.

142. C

Refer to answer above

143. D

Refer to the explanation provided above

144. A

Refer to the explanation provided above

145. E

Refer to the explanation provided above

146. C

147. B, C

148. C

149. D

150. B

151. D

152. A

153. B

154. A, D, E

155. B

156. D

157. D

158. D

159. D

160. A

161. B

162. C

163. C

164. A

165 C

166. D

167. A

Genetics – Questions

168. Frameshift mutation occurs due to?

a. Transition
b. Transversion
c. Insertion
d. Point mutation

169. Chromosome 21 is which type of chromosome?

a. Metacentric
b. Submetacentric
c. Short acrocentric
d. Medium acrocentric

170. Gene silencing RNA-

a. rRNA
b. tRNA
c. miRNA
d. None

171. Y chromosome is?

a. Metacentric
b. Submetacentric
c. Acrocentric
d. Telocentric

172. A mutation in which the base replacement changes the codon for one amino acid to another is called a

a. Missense mutation
b. Nonsense mutation
c. Silent mutation
d. Frame shift mutation

173. Regarding the centimorgan-

a. The human genome is composed of approximately 6000 centimorgans in recombination distance
b. It is a measure of genetic distance that reflects the probability of a crossover between two loci during meiosis
c. Once centimorgan approximates a 5% chance of a crossover during meiosis
d. The average chromosome contains about 500 centimorgans of genetic material

174. Methylation of cytosine (CpG) associated with-

a. Increased expression of protein
b. Decreased expression of protein
c. Point Mutation
d. Change in the function of protein

175. Which of the following disease is X-linked?

a. Thalassemia
b. Color blindness
c. Sickle cell anemia
d. Galactosemia

176. All of the following are autosomal dominant, except?

a. Familial hypercholesterolemia
b. Congenital adrenal hyperplasia
c. Achondroplasia
d. Acute intermittent porphyria

177. Find correct match among the following?

a. Hypophosphatemic rickets-X linked dominant
b. Duchenne muscular dystrophy-X linked recessive
c. Sickle cell: AR
d. Osteogenesis imperfecta-1 : AR
e. Achondroplasia : AR

178. ABO blood group is an example of?

a. Co-dominance
b. AD
c. AR
d. Mitochondrial inheritance

179. Following is transmitted as autosomal dominant disorder-

a. Albinism
b. Sicklecell anemia
c. Hereditary spherocytosis
d. Glycogen storage disease

168. c	169. c	170. c	171. c	172. a	173. b	174. b	175. b	176. b
177. a,b,c	178. a	179. c						

180. Neurofibromatosis I is-
a. AD
b. AR
c. X linked recessive
d. X linked dominant

181. Autosomal dominant inheritance-
a. Albinism
b. Sickle cell anemia
c. Thalassemia
d. Hereditary spherocytosis

182. Inheritance of Huntington's chorea-
a. AD b. XR
c. AR d. XD

183. The inheritance pattern of familial retinoblastoma is-
a. Autosomal recessive
b. Autosomal dominant
c. X-linked dominant
d. X-linked recessive

184. Type of inheritance in tuberous sclerosis-
a. Autosomal dominant
b. Autosomal recessive
c. X-linked dominant
d. X-linked recessive

185. Type of inheritance in Wilson's disease-
a. Autosomal dominant
b. Autosomal recessive
c. X-linked dominant
d. X-linked recessive

186. Hemophilia is associated with-
a. X chromosome
b. Y chromosome
c. Chromosome 3
d. Chromosome 16

187. Which of the following is not X-linked condition?
a. Duchenne muscular dystrophy
b. Emery-Dreifuss muscular dystrophy
c. Facioscapulohumeral muscular dystrophy
d. Becker muscular dystrophy

188. Duchenne muscular dystrophy inherited as-
a. X-linked
b. Autosomal dominant
c. Autosomal recessive
d. Codominant

189. Which of the following is the gene for Duchenne muscular dystrophy?
a. DMPK gene
b. STK11 gene
c. PTCH gene
d. Dystrophin gene

190. Locations of gene which codes for alpha I anti-trypsin deficiency?
a. 14q b. 13p
c. 17p d. 14p

191 Waardenburg syndrome is due to mutation of:
a. PAX2 gene
b. PAX3 gene
c. PAX6 gene
d. PAX9 gene

192. Mutation leading to sickle cell anemia-
a. Crossover mutation
b. Frame shift
c. Deletion
d. Non-disjunction
e. Point mutation

193. The chances of having an unaffected baby, when both parents have Achondroplasia are-
a. 0% b. 25%
c. 50% d. 100%

194. Parents are carrier of an autosomal recessive disorder. Chances of offspring to get affected-
a. 1/1 b. 1/3
c. 1/2 d. 1/4

195. Chance of having cystic fibrosis if only one parent is affected and the other is normal-
a. 25% b. 50%
c. 0% d. 80%

180. a	181. d	182. a	183. b	184. a	185. b	186. a	187. c	188. a
189. d	190. a	191. b	192. e	193. b	194.	195. c		

196. If both parents have sickle cell anemia patients, then the likehood of offsprings having the disease is-

a. 10%
b. 25%
c. 50%
d. 100%

197. Only Males are affected in which of the following pedigree:

a. Autosomal dominant
b. Autosomal recessive
c. Y-linked disorder
d. X-linked recessive

198. Retinitspigmentosa inherited as-

a. AD
b. AR
c. XR
d. Digenic

199. True statement about inheritances of an X-linked recessive trait is-

a. 50% of boys of carrier mother are affected
b. 50% of girls of diseased father are carrier
c. Father transmits disease to the son
d. Mother transmits disease to the daughter

200. Most severe type of Ehlers danlos syndrome?

a. Type 1
b. Type 4
c. Type 8
d. Type 2

201. Osteogenesisimperfecta defect occurs in-

a. Collagen type I
b. Elastin
c. Collagen type IV
d. Basement membrane

202. Hypermobility of joint and hyperelasticity skin is seen in-

a. Marfansyndrome
b. Ehlers-Danlossyndrome
c. Fragile X-syndrome
d. Angelmansyndrome-

203. In Marfan syndrome, the defect in-

a. Fibrillin 1
b. Collagen
c. Fibrillin II
d. Elastin

204. Gene involved in Rett syndrome-

a. P53
b. MECP2
c. RB
d. BRCA

205. The gene that regulates normal morphogeneis during development is-

a. FMR-I gene
b. Homeoboxgene
c. P-16
d. PTEN

206. HOX gene mutation can cause all except-

a. Syndactyly
b. Polydactyly
c. Fused carpal bones
d. VSD

207. Which one of the following is due to point mutation-

a. Color blindness
b. Sickle cell anemia
c. Diabetes
d. Porphyria

208. The following are single gene disorders except-

a. Hemochromatosis
b. Cystic fibrosis
c. William's syndrome
d. Huntington's disease

209. Chromosome involved in friedreisch ataxia:

a. 9q
b. 19q
c. 17p
d. X chromosome

210. Anticipation refers to

a. Waiting for a disease to manifest itself in an individual such as in Huntington's chorea
b. Worsening of a disease phenotype over generations within a family
c. Birth of a fetus with positive prenatal tests for genetic abnormalities
d. Expression of a permutation.

211. Anticipation is seen in-

a. Translocation
b. Chromosome breaking
c. Trinucleotide-repeat expansion
d. Mitochondrial mutation

196. d	197. c	198. b	199. a	200. b	201. a	202. b	203. a	204. b
205. b	206. d	207. b	208. c	209. a	210. b	211. c		

212. True about fragile X syndrome-
a. Triple nucleotide repeat sequence
b. Chromosome breaking
c. Mitochondrial mutation
d. Centromere absent

213. Premutation is seen in:
a. Genomic imprinting
b. Trinucleotide repeat mutation
c. Mitochondrial mutation
d. Gonadal mosaicism

214. All are true about Fragile X syndrome except-
a. Large head
b. Large nose
c. Large ear
d. Large testis

215. One of the following disorders is due to maternal disomy of chromosome 15-
a. Prader-Willisyndrome
b. Angelmansyndrome
c. Hydatidiform mole
d. Klinefelter's syndrome

216. True about genomic imprinting-
a. Differential expression of gene depending on parent of origin
b. Prader-Willi syndrome is maternal deletion of chromosome 15
c. Angelman syndrome is due to paternal deletion of chromosome 15
d. Uniparentaldisomy is other name of genomic imprinting

217 These syndromes are associated uniparentaldisomy except-
a. Angelman
b. Praderwillisyndrome
c. Russel silver
d. Bloom Syndrome

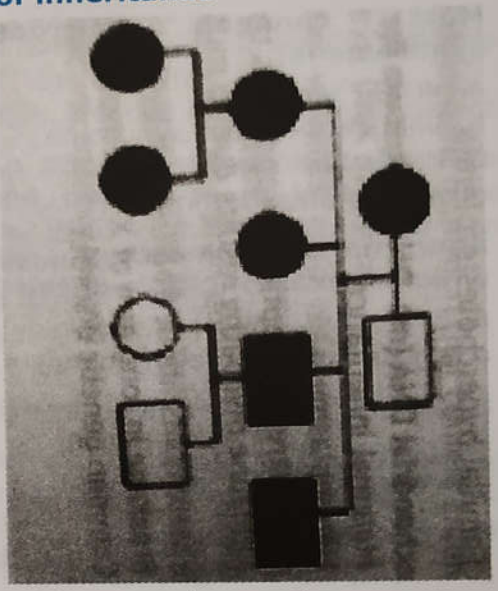
218. Paternal 15 chromosome deletion is seen in-
a. Angelmansyndrome
b. Prader-Willisyndrome
c. Down syndrome
d. Turner syndrome

219. Angelman syndrome is due to-
a. Digenicinheritance
b. Inversion
c. Uniparentaldisomy
d. Mitochondrialdisorder

220. Mitochondrial chromosomal abnormality leads to-
a. Lebershereditaryopticneuropathy
b. Angelmansyndrome
c. Prader-Willi syndrome
d. Myotonicdystrophy

221. Type of inheritance in Melas-
a. AD
b. AR
c. Mitochondrial
d. X-linked

222. Pedigree analysis-analyze the following pedigree and give the mode of inheritance-



a. Autosomal recessive
b. Autosomal dominant
c. Mitochondrial inheritance
d. X linked dominant

223. False statements about fragile X syndrome is-
a. Breakage in long arm of X chromosome
b. Common genetic disorder
c. Micro-orchidism
d. Long face

224. Which chromosome is responsible for the production of MIF?
a. Chromosome 16
b. Chromosome 22
c. X Chromosome
d. Y Chromosome

212. a	213. b	214. b	215. a	216. a	217. d	218. b	2191. c	220. a
221. c	222. c	223. c	224. b					

225. Velocardiofacial defect is due to mutation in?
a. Chr 11 b. Chr 13
c. Chr 22 d. Chr 3

226. Cri du chat syndrome is?
a. 22q b. 5q
c. 22p d. 5p

227. If a chromosome divides in an axis perpendicular to usual axis of division it is going to form:
a. Ring chromosome
b. Isochromosome
c. Acrocentric chromosome
d. Subtelocentric chromosome

228. Most common microdeletion syndrome is?
a. WAGR syndrome
b. Velo-cardio-facial syndrome
c. PraderWilli syndrome
d. Angelman syndrome

229. Abnormal chromosomal number which is exact multiple of the 23 is-
a. Euploidy b. Aneuploidy
c. Mosaicism d. Trisomy

230. Edward's syndrome is-
a. Trisomy 21 b. Trisomy 18
c. Trisomy 13 d. 5P

231. Patau's syndrome is due to-
a. Trisomy 21 b. Trisomy 18
c. 45Xo d. Trisomy 13

232. Down's syndrome increases risk of: (PGI)
a. Hirschprung's disease
b. Leukemia
c. Sensorineural hearing loss
d. Hyperthyroidism
e. Atlanto-occipital dislocation

233. Chromosomal abnormalities in Down syndrome is/are due to: (PGI)
a. Nondisjunctional of maternal chromosome
b. Nondisjunctional of paternal chromosome
c. Translocations between chromosome 21 & 14
d. Disjunction of paternal chromosome
e. Mosaicism

234. True about Lyonization of X chromosome:
a. Inactivation of X chromosome only in somatic cell
b. Inactivation of X chromosome only in germ cell
c. Inactivation of X chromosome in somatic & germ cell both
d. Maximum number of Barr body is equal to X chromosome

235. Which chromosome contains the most known genetic disease of any human chromosome?
a. 1 b. 9
c. 11 d. 6

236. Down syndrome is most commonly caused by-
a. Maternalnondisjunction
b. Paternalnondisjunction
c. Translocation
d. Mosaicism

237. Down syndrome is due to-
a. Trisomy 21
b. Translocation 13-15/21
c. Translocation 22/21
d. All of the above

238. Which of the following is not associated with Down syndrome?
a. Trisomy 21
b. Mosaic 21
c. Translocation t (15,21), t (22,21)
d. Deletion of 21

239. All of the following are characteristic of Turner syndrome except-
a. Webbing of neck
b. Cubitus valgus
c. Umbilical hernia
d. Coarctation of aorta

240. Blood group antigen A and B are associated with which chromosome-
a. Chromosome 1
b. Chromosome 5
c. Chromosome 9
d. Chromosome 19

225. c	226. d	227. b	228. b	229. b	230. b	231. d	232. a,bc,e
233. a, c,e	234. c	235.	236. a	237. d	238. d	239. c	240. c

241. Karyotype in Klinefelter's syndrome is-

a. 47XXY
b. 46XXY
c. 445XO
d. 45XXX

242. Which of the following is not the karyotype of Turner syndrome?

a. 46, XX, i (Xq)
b. 45, XO
c. 46, XX, I (X p)
d. 46, XX, r (X)

243. A nineteen-year old female with short stature, widespread nipples and primary amenorrhoea most likely has a karyotype of-

a. 47, XX + 18
b. 46, XXY
c. 47, XXY
d. 45 X

244. Males who are sexually underdeveloped with rudimentary testes and prostate glands, sparse pubic and facial hair, long arms and legs and large hands and feet are likely to have the chromosome-

a. 45, XYY
b. 46, XY
c. 47, XXY
d. 46, X

245. Streak gonads are seen in-

a. Turner syndrome
b. Klinefelter'ssyndrome
c. Patau'ssyndrome
d. Down syndrome

246. Barr body is absent in-

a. Klinefelter's syndrome
b. Turner's syndrome
c. Super female
d. None

247. Condition not associated with increased risk of cancer are all except?

a. NF-1
b. Turners syndrome
c. Down syndrome
d. Chr 13 deletion

248. A child with a small head, minor anomalies of face including a thin upper lip, growth delay and development disability can have all of the following except-

a. A chromosomal syndrome
b. A teratogenic syndrome
c. A mendelian syndrome
d. A polygenic syndrome

249. Which of the following is true regarding ataxia telanglectasia?

a. It is X linked recessive disease
b. It is autosomal recessive disorder
c. It is associated with increased levels of IgM
d. None of the above

250. Xerodermapigmentosa all true except:

a. Autosomal dominant
b. Onset of squamous cell ca by 2^{nd}-3^{rd} decade
c. Develops non melanotic cancer by the age of 9 years
d. Nucleotide excision repair defect

251. An obese women with T2 diabetes and hypertension had endometrial ca. Which of the following gene is involved?

a. PTEN
b. P53
c. B CATENIN
d. SMAD

252. BRCA1 is the most common gene mutated in familial breast cancer. This gene is located on which chromosome?

a. 13
b. 17
c. 20
d. 21

253. BRCA1 and BRCA2 is responsible for all these Cancers except:

a. Breast
b. Ovary
c. Intestinal lining cancer
d. Prostate

254. Multifactorial inheritance is most likely to play a significant role in the appearance of-

a. Achondroplasia
b. Lysosomal storage disease
c. Cleft lip
d. Huntington disease

241. a	242. c	243. d	244. c	245. a	246. b	247. d	248. d
249. b	250. a	251.	252. b	253. c	254. c		

255. Which of the following is a DNA repair defect?

a. Bloom syndrome
b. Incontinentiapigmenti
c. Aplastic anemia
d. Tuberous sclerosis

256. Obesity with mental retardation is seen in:

a. Prader-Willisyndrome
b. Turner'ssyndrome
c. Fragile X syndrome
d. Noonamsyndrome

257. Multifactorial inheritance is seen in:

a. Neurofibroma
b. Hemophilia
c. Cardiac septal defects
d. Hypophosphatemic rickets

258. The best suited nucleated cell for chromosomal study-

a. Polymorphs
b. Lymphocytes
c. Epithelial cells
d. Langerhan's cell

259. Which of the following not used to study epigenetics?

a. Chip on chip
b. Bisulfite sequencing
c. HPLC
d. Methylation-sensitive restriction enzymes

260. Which of the following procedures as routine technique for karyotyping using light microscopy?

a. C-banding
b. G-banding
c. Q-banding
d. Ethidium bromide staining

261. Karyotyping of the fetus is done in all except-

a. Chorionic villi sampling
b. Cordocentesis
c. Amniocentesis
d. Fetal skin biopsy

262. Microarray is-

a. Study of multiple genes
b. Study of disease
c. Study of organisms
d. Study of blood group

263. Chromosomes are visualised through light microscope with resolution of-

a. 5kb
b. 50mb
c. 5mb
d. 500kb

264. Karyotyping is done in which phase of cell cycle-

a. Anaphase
b. Metaphase
c. Telophase
d. S phase

265. Feulgen reaction is a cytochemical test for-

a. Mrna
B. Trna
c. DNA
d. All of the above

255. a	256. a	257. c	258.	259. b	260. b	261. d	262. a
263. a	264. b	265. b					

168. C

A transition is a point mutation that replaces a purine-pyrimidine base pair with a different purine-pyrimidine base pair. For example, an A-T base pair becomes a G-C base pair.

A transversion is a point mutation that replaces a purine-pyrimidine base pair with a pyrimidine-purine base pair. For example, an A-T base pair becomes a T-A or a C-G base pair.

Frameshift/in-frame: addition or deletion of base(s) - Usually nonfunctional; often shorter than normal

Frameshift Mutation (1bp deletion)
ATGGCAATTCGTTTTTACCTATAGGG... DNA coding strand
Met Ala Ile Arg Phe Tyr Leu Stop
Amino Acid

169. C

Chromosome 21 is Short-Acrocentric. This method of classification is not used now-a-days.

Translocation between two acrocentric chromosomes leads to Down's syndrome

Chromosomes 13,14,15 (medium acrocentric), 21,22,Y (Short scrocentric)

170.C

miRNA

The miRNAs do not encode proteins; instead, they function primarily to modulate the translation of target mRNAs into their corresponding proteins.

Posttranscriptional silencing of gene expression by miRNA is a fundamental and well-conserved mechanism of gene regulation present in all eukaryotes (plants and animals).

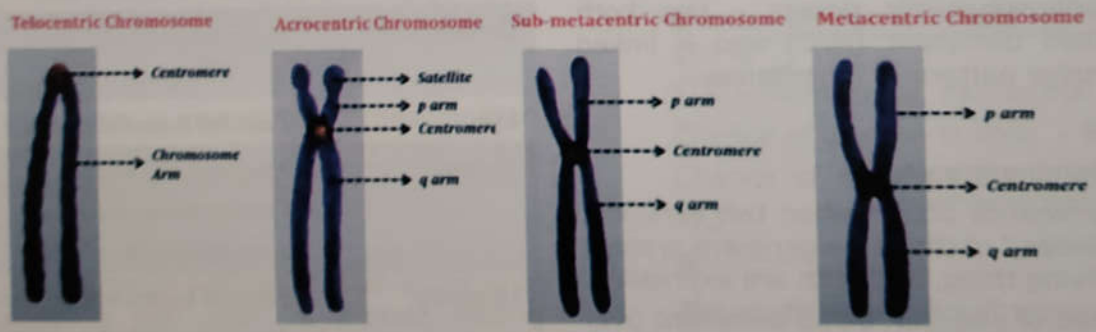
171.C

Chromosomes.

Acrocentric. If the p (short) arm is so short that it is hard to observe, but still present, then the chromosome is acrocentric

The human genome includes five acrocentric chromosomes: 13, 14, 15, 21, 22 and Y

X chromosome is sub-metacentric



172.A

Mutation

Silent: new codon specifies same amino acid

Missense: new codon specifies different amino acid

Nonsense: new codon is stop codon

173.B

CentiMorgan (cM)

The recombination frequency provides a measure of genetic distance between any

pair of linked loci. is distance is expressed in centimorgans.

One centimorgan is equal to 1% recombination frequency/ crossing over

1 cM is approximately equal to 1 million base pairs of DNA (1 Mb)

Female genome is approx 4500 cM long and male genome is approx. 3000 cM long

174. B

DNA Methylation

DNA methylation is an epigenetic mechanism that occurs by the addition of a methyl (CH3) group to DNA, thereby often modifying the function of the genes and affecting gene expression.

DNA methylation process is the covalent addition of the methyl group at the 5-carbon of the cytosine ring resulting in 5-methylcytosine (5-mC), also informally known as the "fifth base" of DNA.

These methyl groups project into the major groove of DNA and inhibit transcription.

175. B

176. B

177. A, B, C

Hypophosphatemic rickets – Has both X-linked dominant (M/C) and X linked recessive pattern of inheritance.

178. A

Co-Dominant pattern.

Codominance occurs when two versions, or "alleles," of the same gene are present in a living thing, and both are expressed.

Instead of one trait being dominant over the other, both traits appear

Eg., ABO blood group and MHC are co-dominant.

Let's see AB blood group – where A and B are dominant alleles but both of them manifest in AB blood group

179. C

180. A

181. D

182. A

183. B

184. A

185. B

186. A

187. C

Facioscapulohumeral muscular dystrophy

FSHD1 is inherited in an autosomal dominant pattern

FSHD2 is inherited in a digenic pattern – which involves mutation in two genes

188. A

For all the above questions on inheritance – There are few must know inheritance patterns below. Try to read them and you will know more inheritance as you read individual diseases

Autosomal Dominant

System	Disorders Disorder
Nervous	Huntington disease Neurofibromatosis Myotonic dystrophy Tuberous sclerosis
Urinary	Polycystic kidney disease (AD)
Gastrointestinal	Familial polyposis coli
Hematopoietic	Hereditary spherocytosis von Willebrand disease
Eye	Familial Retinoblastoma
Skeletal	Marfan syndrome Osteogenesisimperfecta Achondroplasia
Metabolic	Familial hypercholesterolemia Acute intermittent porphyria

Autosomal Recessive Disorders

System	Disorders
Metabolic	Cystic fibrosis Phenylketonuria Galactosemia Homocystinuria α1-Antitrypsin deficiency Wilson disease Hemochromatosis
Hematopoietic	Sickle cell anemia, Thalassemias

Endocrine	Congenital adrenal hyperplasia
Skeletal	Ehlers-Danlos syndrome (some variants) Alkaptonuria
Nervous	Neurogenic muscular atrophies Friedreich ataxia Spinal muscular atrophy

X-Linked recessive disorders

System	Disease
Musculoskeletal	Duchenne muscular dystrophy
Blood	Hemophilia A and B, Chronic granulomatous disease (membrane NADPH oxidase defect) Glucose-6-phosphate dehydrogenase deficiency
Eye	Colour blindness
Immune	Agammaglobulinemia, Wiskott-Aldrich syndrome
Metabolic	Diabetes insipidus, Lesch-Nyhan syndrome
Nervous	Fragile X syndrome

189. D. Dystrophin gene.

DMD

Duchenne and Becker muscular dystrophy are caused by loss-of-function mutations in the dystrophin gene on the X chromosome.

Dystrophin is one of the largest human genes, spanning 2.3 million base pairs and composed of 79 exons

190. A

α_1-Antitrypsin deficiency is an autosomal recessive dis- order of protein folding marked by very low levels of circulating α_1-Antitrypsin (α_1AT)

α_1AT is a small 394–amino acid plasma glycoprotein synthesized predominantly by hepatocytes

The gene, located on chromosome 14,

The patients develop liver cirrhosis and pan acinar type of emphysema.

191. B

Waardenburg syndrome is a group of genetic conditions that can cause hearing loss and changes in coloring (pigmentation) of the hair, skin, and eyes

Type III (sometimes called Klein-Waardenburg syndrome) includes abnormalities of the arms and hands in addition to hearing loss and changes in pigmentation

Klein-Waardenburg syndrome (WS-III) is one such developmental disorder resulting from a mutation in a PAX 3 gene

192. E

Sicklecellanemia

Mutation in sicklecellanemiais a point mutation

It's a mutation in the beta globinchain in the 6th position – which changes glutamine to valine.

The nextfewquestions are on recurrentriskcalculation. Follow the basicrules for solving the problems.

AD – Oneparentaffected – chance of diseasein offsprings – 50%

AD – Bothparentsaffcted – chance of disease inoffsprings – 75%

AR – Bothparentscarriers - chance of diseasein offsprings – 25%

AR – Oneparentisaffected, otherparentisnormal

chance of disease in offspring – 0%

chance of carrier state in offspring – 100%

XLR – Momcarrier, dadnormal

Chance of disease in sons - 50%

Chance of carrier state in daughters – 50%

193. B

Achodroplasiaisautosomaldominant.

So as per the pointsdiscussedabove, the riskwill be – 25% (unaffectedkid)

194. ¼

Autosomal recessive inheritance the chance of the offspring having the disease is 1/4

195. C

Cystic fibrosis is autosomal recessive disease.

So as per the pointsdiscussedabove, the riskwill be – 0% (unaffectedkid)

196. D

Sickle cell anemia is autosomal recessive.

Both parents are having the disease → Means both parents are having both alleles mutated.

So the offsprings will get one affected allele from each parent – So all offsprings will be affected

197. C

The best answer here will be Y linked disorder.

In XLR also males are predominantly affected. Females having the disease in XLR – is very very rare. (but possible), so the best answer here will be Y linked

198. AR

Retinitis Pigmentosa.

Retinitis pigmentosa isinheritedas AD, AR and X linked. So wehave to go with most common mode asanswer for the question.

This question has various answers. Standard textbookalsovaries. But american academy of opthalmologygoes with AR, so we are going with AR as the most common mode of inheritance

199. A

Just see the rules of inheritance in the beginning of the session.

200. B

Most sever type ofEhlerdanlossyndrome is Type 4

Type 4 is vascular type ofEhlerDanlosSyndrome.

Type 4 – causesaorticdissection and death, thusmaking it the most severe type.

201. A

OsteogenesisImperfecta

Osteogenesisimperfecta (*OI*), also known as brittle bone disease, is a group of genetic disorders that mainly affect the bones.

Osteogenesisimperfecta (*OI*) is a disorder of bone fragility chiefly caused by mutations in the COL1A1 and COL1A2 genes that encode type I collagen

202. B

Ehlers-Danlos syndrome (EDS) is a group of inherited connective tissue diseases that have in common a defect in collagen structure or synthesis. Clinically, the disease causes hyperextensible skin that is easily traumatized and hyperextensible joints secondary to effects on the joints and adjacent ligaments.

Types of RDS

Kyphoscoliotic EDS: autosomal recessive form

Vascular variant EDS: autosomal dominant form that causes rupture of vessels and bowel wall

Classical EDS: autosomal dominant form that causes a type V collagen defect; patients have a normal lifespan

203. A

Marfan syndrome

Marfan syndrome is due to a mutation of the fibrillingene (*FBN1*) on chromosome 15q21.

Fibrillin is a glycoprotein that functions as a scaffold for the alignment of elastic fibers.

204. B

Rett Syndrome

Rett syndrome is a neurodevelopmental disorder that affects girls almost exclusively.

It is characterized by normal early growth and development followed by a slowing of development, loss of purposeful use of the hands, distinctive hand movements, slowed brain and head growth, problems with walking, seizures, and intellectual disability

Rett syndrome are caused by a mutation in the methyl CpG binding protein 2, or *MECP2* gene

205. B

HOX genes.

The regulatory proteins themselves are encoded by genes called homeobox(HOX) or homeotic genes. Another closely related set of genes is the PAX (paired-box) genes.

Sequential and coordinated gene

expression is necessary for proper tissue and cell differentiation during embryonic life

206. D

Hox genes mutations generally cause abnormalities in the limb orientation and differentiation

There are more than 35 different syndromes which result in mutation of various HOX genes, most of them will have limb abnormalities

207. b

Sicklecellanemia

Mutation in sicklecellanemiais a point mutation

It's a mutation in the beta globinchain in the 6th position – which changes glutamine to valine

208. C

William syndrome.

It's not a single gene disorder.

Most cases of Williams syndrome are not inherited but occur as random events during the formation of reproductive cells (eggs or sperm) in a parent of an affected individual. These cases occur in people with no history of the disorder in their family.

Williams syndrome is considered an autosomal dominant condition because one copy of the altered chromosome 7 in each cell is sufficient to cause the disorder.

209. A

Freidriech Ataxia

Mutations in the FXN gene cause Friedreich ataxia. This gene provides instructions for making a protein called frataxin

The gene is located in chromosome 9q

Patients develop impaired muscle coordination (ataxia) that worsens over time

210. B

Anticipation

The signs and symptoms of some genetic conditions tend to become more severe and appear at an earlier age as the disorder is passed from one generation to the next.

Occurs with disorders that are caused by an unusual type of mutation called a trinucleotide repeat expansion.

The pathogenesis is dynamic here and it the repeats expand during gametogenesis.

211. C (See above question)

212. A

Fragile X syndrome is a genetic condition that causes a range of developmental problems including learning disabilitie and cognitive impairment

Fragile X is because of CGG repeats in FMR1 gene.

It has XLR inheritance pattern.

Macro-orchidism is one of the salient features

213. B

Premutation

A premutation is a situation in which there are an excess number of repeats in a gene that is at risk of increasing in length during reproduction but which does not cause disease in the person with the excess number of repeats

Seen in trinucleotide repeat disorders.

214. B

Features of Fragile X syndrome

Affected males are *mentally retarded*, with an IQ in the range of 20 to 60.

They express a characteristic physical phenotype that includes a *long face with a large mandible, large everted ears*, and *large testicles (macro-orchidism)*.

Hyperextensible joints, a high arched palate, and mitral valve prolapse

215. A

Prader- Willi syndrome

Deletion of band q12 on long arm of paternal chromosome 15 occurs or maternal disomy of Ch 15

Genes in this region of maternal chromosome 15 are imprinted so there is complete loss of their functions.

Patients have mental retardation, short

stature, hypotonia, hyperphagia, small hands and feet, and hypogonadism.

216. A

Imprinting

Imprinting involves transcriptional silencing of the paternal or maternal copies of certain genes during gametogenesis.

For such genes, only one functional copy exists in the individual. Loss of the functional (not imprinted) allele by deletion gives rise to diseases.

217. D.

Examples for imprinting disorders

Angelmansyndrome

Praderwillisyndrome

Silver Russel syndrome

Beckwithwidemansyndrome

Pseudo-Hypoparathyroidism

218. B

Prader- Willi syndrome

Deletion of band q12 on long arm of paternal chromosome 15 occurs or maternal disomy of Ch 15

219. C

Inheritance of both chromosomes of a pair from one parent is called *uniparentaldisomy*

Angelman syndrome, can result from uniparentaldisomy of paternal chromosome 15.

220. A

Leber hereditary optic neuropathy is a prototype of mitochondrial disorder.

It is a neurodegenerative disease that manifests as a progressive bilateral loss of central vision.

Visual impairment is first noted between ages 15 and 35, leading eventually to blindness.

221. C

Mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS) it's a mitochondrial inherited disorder

222. C

In the above pedigree

When mom is affected all kids are affected

When dad is affected none are affected

This is classical of mitochondrial inheritance.

223. C

Fragile X patients express a characteristic physical phenotype that includes a *long face with a large mandible, large everted ears*, and *large testicles (macro-orchidism).*

224. B

MIF – Macrophage Migration Inhibitory factors is located in Chromosome 22.

Mutation in this is known to be associated with Juvenile Rheumatoid arthritis

225. C. Chromosome 22

Velocardio facial syndrome

Chromosome 22q11.2 deletion syndrome encompasses a spectrum of disorders that result from a small deletion of band q11.2 on the long arm of chromosome 22.

Clinical features include *congenital heart defects, abnormalities of the palate, facial dysmorphism, developmental delay, and vari- able degrees of T-cell immunodeficiency and hypocalcemia.*

Previously, these clinical features were considered to rep- resent two different disorders—*DiGeorge syndrome* and *velocardiofacial syndrome*.

226. D. 5p

Cri Du chat Syndrome.

Deletion of chromosome 5p

High pitched cry

Congenital heart defects

Mental retardation, microcephaly

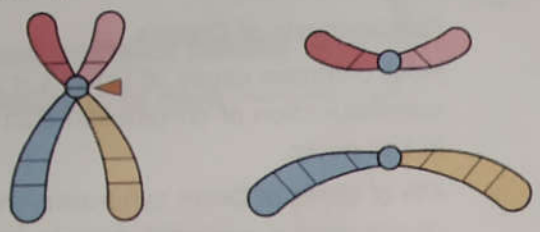
Can be diagnosed by FISH and Karyotyping

227. B – Isochromosome

An isochromosome has morphologically identical genetic information in both arms.

The most common isochromosome present in live births involves the long arm of the X and is designated *i(X)(q10)*

It is as a result of the plane of separation being horizontal than vertical.



228. B

Microdeletion syndromes.

Usually involves less than 5Mb base pairs

Most common micro-deletion syndromes isDigeorge (Velocardiofacial syndrome)

Microdeletion syndromes are diagnosed by FISH

229. B

Any exact multiple of the haploid number of chromosomes (23) is called *euploid.*

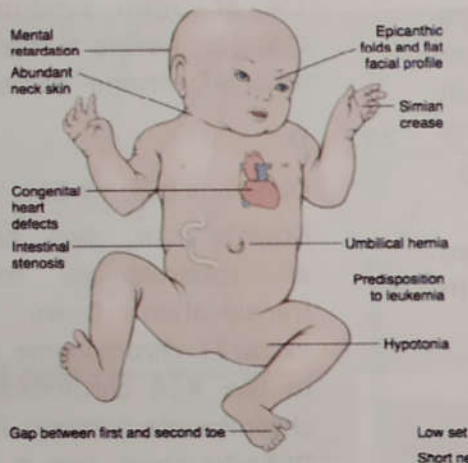
If an error occurs in meiosis or mitosis and a cell acquires a chromosome complement that is not an exact multiple of 23, it is referred to as *aneuploidy.*

230. B

Trisomy 18: Edwards syndrome

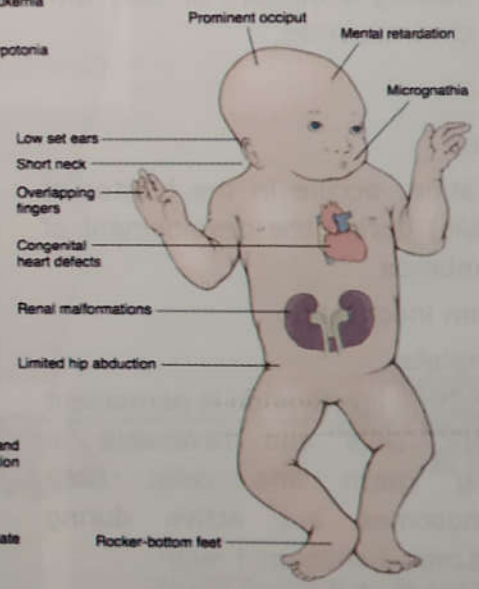
231. D

Trisomy 13 – Patau Syndrome



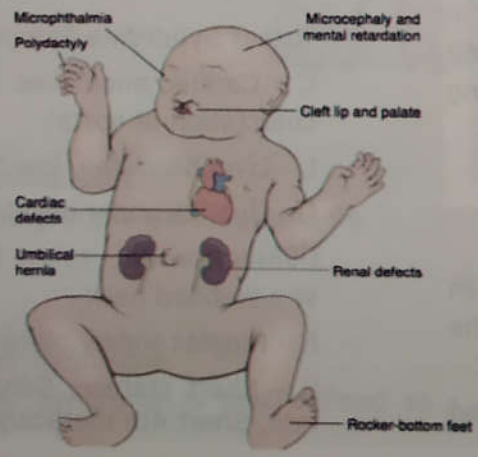
TRISOMY 21: DOWN SYNDROME

Incidence: 1 in 700 births
Karyotypes:
Trisomy 21 type: 47,XX, +21
Translocation type: 46,XX,der(14;21)(q10;q10),+21
Mosaic type: 46,XX/47,XX, +21



TRISOMY 18: EDWARDS SYNDROME

Incidence: 1 in 8000 births
Karyotypes:
Trisomy 18 type: 47,XX, +18
Mosaic type: 46,XX/47,XX, +18



TRISOMY 13: PATAU SYNDROME

Incidence: 1 in 15,000 births
Karyotypes:
Trisomy 13 type: 47,XX, +13
Translocation type: 46,XX,+13,der(13;14)(q10;q10)
Mosaic type: 46,XX/47,XX, +13

232. A, B, C, E

Thyroid and Down's

About half of the patients with Down syndrome have been shown to have subclinical hypothyroidism with elevated TSH and normal thyroxine levels.

Hyperthyroidism is much less frequent in patients with Down syndrome as compared to hypothyroidism

233. A,C,E

Pathogenesis of Down's

Most common cause of Down's is meiotic nondisjunction of chromosome 21 occurs in the ovum.

4% of cases of Down syndrome, the extra chromosomal material derives from the presence of a rob- ertsonian translocation of the long arm of chromosome 21 to another acrocentric chromosome (e.g., 22 or 14)

1% of Down syndrome patients are mosaics, having a mixture of cells with 46 or 47 chromosomes.

234. C

Lyonisation

X inactivation, occurs in the blastocyst (~100 cells) during the development of female embryos

It's random inactivation

It's incomplete

X-chromosome inactivation is permanent in somatic cells and reversible in developing germ line cells. Both X chromosomes are active during oogenesis.

235. 1

Chromosome 1

Chromosome 1 is the largest human chromosome, containing about 8% of the entire genome

The chromosome carries an estimated 3,141 genes

More than 350 diseases linked to chromosome 1

236. A

Pathogenesis of Down's

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1% of Down syndrome patients are mosaics, having a mixture of cells with 46 or 47 chromo- somes

237. D

Down's is trisomy 21.

Down's can also be as a result of robertsonian translocation. In translocation Down syndrome, the extra 21 chromosome may be attached to the #14 chromosome, or to other chromosome numbers like 13, 15, or 22. In some cases, two # 21 chromosomes can be attached to each other

238. D

Deletion of 21 chromosome will not result in Down's syndrome.

239. C (Best option in this MCQ)

Turner Syndrome Symptoms - CLOWNS

C - Cardiac anomalies (most common - coarctation of aorta)

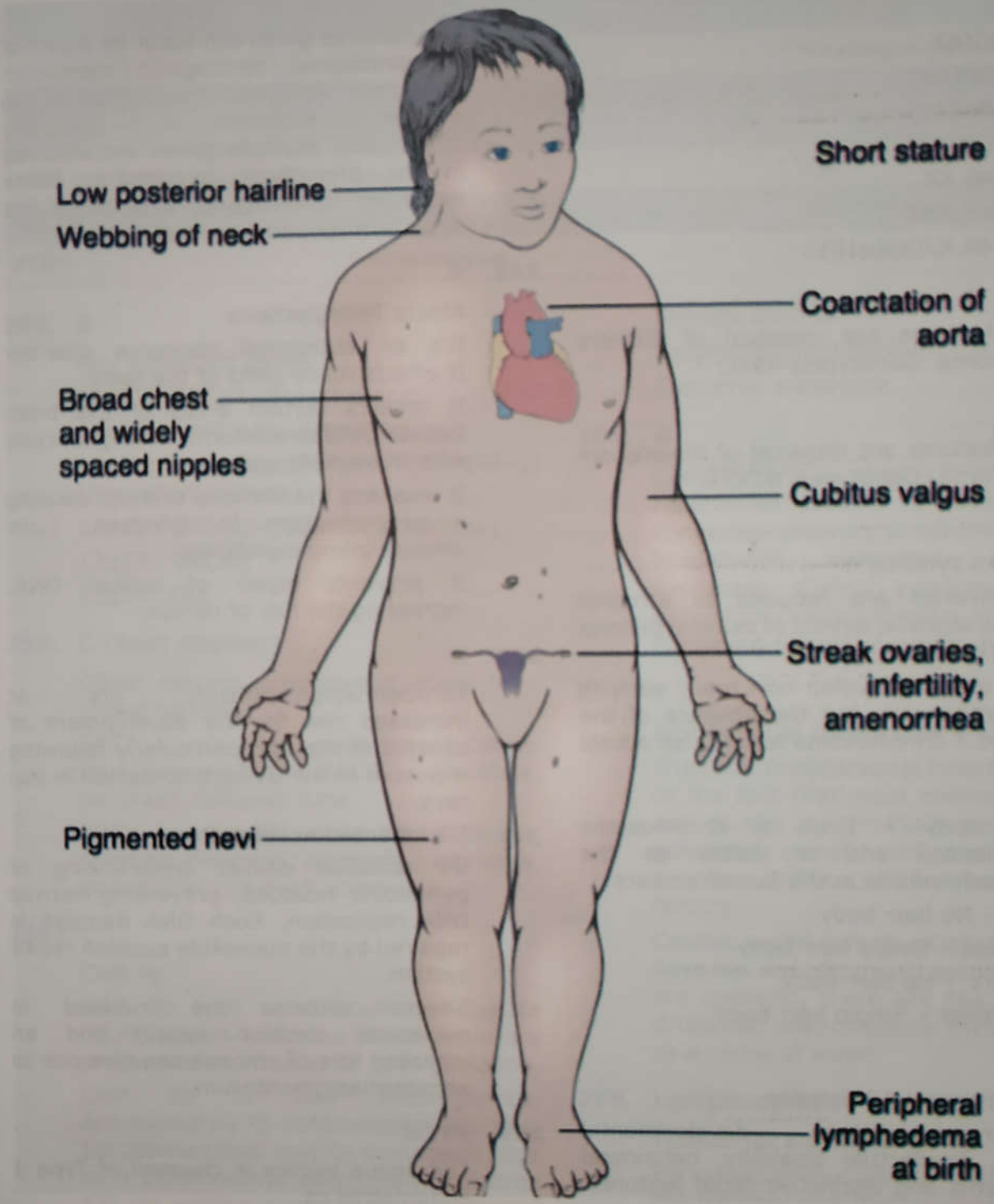
L - Lymphoedema, low thyroid

O - Ovaries under developed (streak ovaries)

W - Webbed neck

N - Nipples widely placed

S - Short stature, Sensoneural hearing loss, Short 4th metacarpal



240. C

Chromosome location

ABO blood group – Chromosome 9q

Rh blood group – Chromosome 1

241. A

Klinefelter Syndrome

Klinefelter syndrome is best defined as male hypogonad- ism that occurs when there are two or more X chromo- somes and one or more Y chromosomes

The incidence of this condition is approximately 1 in 660 live male births

Chromosome – 47 XXY

242. C

TurnersSyndrome –

Chromsomalabnormalities (Apartfrom 45XO)
46,X,i(Xq)
46,X,del(Xq)
46,X,del(Xp)
46,Xr(X)
45,X/46,XX
45,X/47,XXX
45,X/46,X,i(X)(q10)

243. D

The features are classical of Turners Syndrome. Genotypeis 45XO

244. C

The features are classical of Klinefelters Syndrome. Genotypeis 47XXY.

245. A

Turners syndrome

The ovaries are reduced to atrophic fibrous strands, devoid of ova and follicles *(streak ovaries)*

Fetal ovaries develop nor- mally early in embryogenesis, but the absence of the second X chromosome leads to an accele

246. B

Barr body – Extra X chromosome is silenced and is visible as the heterochromatin in the buccal smears

Male – No barr body

Females – Single barr body

Turners - No barr body

Klinefelter – Single barr body

247. D

Chromosome 13 deletion

Features include developmental delay, intellectual disability, behavioral problems and distinctive facial features. There is no increase in risk of malignancy

248. D

The features given can occur as a part of a chromosomal / teratogenic / mendelian syndrome. Polygenic inheritance is the answer of exclusion. In a polygenic inheritance, multiple genes are involved in the phenotypic expression. Some examples of polygenic inheritance are hypertension and diabetes.

249. B

Ataxia Telangiectasia

It's an autosomal recessive disorder It affects many parts of the body:

It impairs certain areas of the brain including the cerebellum, causing difficulty with movement and coordination.

It weakens the immune system, causing a predisposition to infection. (Low amount immunoglobulins)

It prevents repair of broken DNA, increasing the risk of cancer.

250. A

Xerodermapigmentosum, are at increased risk for the development of cancers of the skin particularly following exposure to the UV light contained in sun rays.

It's inherited as AR pattern

UV radiation causes cross-linking of pyrimidine residues, preventing normal DNA replication. Such DNA damage is repaired by the nucleotide excision repair system.

Several proteins are involved in nucleotide excision repair, and an inherited loss of any one can give rise to xerodermapigmentosum

251. PTEN

The above history is classical of Type 1 endometrial ca.

Features	Type 1	Type 2
Prototypic form	Endometrioid carcinoma	Uterine (papillary) serous carcinoma
typical patient	Perimenopausal or early postmenopausal women	Elderly women
	Background of endometrial hyperplasia	Background of atrophic endometrium

	Low-grade	High-grade
	Estrogen-dependent	Not estrogen-dependent
Estrogen receptor	usually positive; high grade cases may be negative	negative
MIB1 proliferation index (ki-67)	low	high
p53	negative; high grade cases may be positive	diffuse positivity
PTEN	Positive mutation	No mutation

252. B

Few important genes

17q21 : BRCA1 (Breast cancer)

17q12 : Her2/neu (Breast cancer)

17q11: Neurofibromin (NF-1) (Neurofibromatosis)

17p13 : p53 (Li-Fraumeni syndrome)

13q12 : BRCA2

13q14: RB1

253. C (Best answer)

Other cancers predisposed apart from breast cancer.

BRCA 1 - Ovarian, male breast cancer (but lower than *BRCA2*), prostate, pancreas, fallopian tube

BRCA 2 - Ovarian, male breast cancer, prostate, pancreas, stomach, melanoma, gallbladder, bile duct, pharynx

254. C

Cleft lip

Cleft lip with or without cleft palate (CL/P) occurs when the frontal maxillary process fails to fuse at 35 days gestation.

Cleft lip is very heterogenous. Approximately 75-80% cases are sporadic. Syndromic cases may be monogenic, due to a chromosome abnormality (trisomy 13), or environmental factors (maternal rubella). Other cases are familial

255. A

Below are a list of DNA repair defect syndromes

Ataxia telangiectasia

Nijmegen breakage syndrome

Werner syndrome

Bloom syndrome

Fanconi anemia

Xerodermapigmentosum

Cockayne syndrome

256. A

Constitutional obesity and mental retardation cooccur in several multiple congenital anomaly syndromes, including Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, Albright hereditary osteodystrophy, and Borjeson-Forssman-Lehmann syndrome

257. C

Multifactorial Inheritance

The term multifactorial inheritance refers to the fact that most common diseases are caused by multiple genes (i.e., a polygenic component) and expression is often influenced by environmental factors.

Cardiac septal defects – Only a quite a few have few specific genes associated. Most are sporadic, there are few teratogenic drugs and environmental toxins proposed as a cause in some.

258. Lymphocytes

Tissues for karyotyping

Peripheral venous blood - most commonly, the lymphocytes (best source)

Skin (fibroblasts), bone marrow.

For fetal chromosome patterns - amniotic fluid cells, chorionic villi.

5-10 mL of heparinized venous blood is the most commonly used source.

259. C

Epigeneticstudies

Epigenetic research includes chromatin

immunoprecipitation(Chip-on-chip and ChIP-Seq), fluorescent in situ hybridization, methylation-sensitive restriction enzymes, DNA adenine methyltransferase identification (DamID) and bisulfite sequencing

260. B

Karyotype Staining

A variety of staining methods have been developed that allow identification of individual chromosomes on the basis of a distinctive and reliable pattern of alternating light and dark bands.

The one most commonly used involves a Giemsa stain and is hence called *G banding*.

261. D

Fetal skin biopsy is not used for karyotyping, because the the fibroblasts mature enough to do karyotyping studies only after 2nd trimester. So the purpose of inutero diagnosis is lost.

So fetal skin biopsy is not useful for karyotyping.

262. A

Micro Array – Studies all genes known.

In array CGH, the test DNA and a reference (normal) DNA are labeled with two different fluorescent dyes.

The differentially labeled samples are then co- hybridized to an array spotted with DNA probes that span the human genome at regularly spaced intervals, and usually cover all 22 autosomes and the sex chromosomes.

263. 5 Mb

Resolution.

Conventional karyotyping is limited to the detection of rearrangements involving more than 5 Mb of DNA.

The resolution of the FISH technique, using fluorescent probes, is about 100kb-1Mb in size.

264. B

Karyotyping

The study of chromosomes— *karyotyping*

The usual procedure to examine chromosomes is to arrest dividing cells in metaphase with mitotic spindle inhibitors (e.g., *N*-diacetyl-*N*-methylcolchicine [Colcemid]) and then to stain the chromosomes.

265. DNA

Feulgen Reaction.

Used in histology to identify chromosomal material or DNA in cell specimens.

It is darkly stained. It depends on acid hydrolysis of DNA, therefore fixating agents using strong acids should be avoided.

Diseases of the Immune System – Questions

266. Interleukin secreted by Th1 cells include?

a. IL-2 b. IL-4
c. IL-10 d. IL-13

267. Castleman syndrome is associated with?

a. EBV b. CMV
c. HHV8 d. Herpes virus

268. Which of the following cells have MHC class II?

a. RBC
b. B cells, Dendritic cells, NK cells
c. All nucleated cells
d. Platelets

269. During development, all the antigens of self are introduced to thymic cells in-order to be removed to prevent autoimmunity. Which of the following genes is involved in the process?

a. NOTCH1 b. AIRE
c. BB gene d. CPK gene

270. Antibody in diffuse scleroderma:

a. Anti-DNA topoisomerase
b. Anti-Centromere
c. Anti-RNA polymerase
d. Anti-Smith

271. Class IV Lupus nephritis-

a. Mesangial lupus nephritis
b. Proliferative lupus nephritis
c. Membranous lupus nephritis
d. Diffuse sclerosing lupus nephritis

272. Most severe form of lupus nephritis:

a. Mesangial lupus lomerulonephritis
b. Focal proliferative glomerulonephritis
c. Diffuse proliferative glomerulonephritis
d. Membranous glomerulonephritis

273. Speckled pattern is seen in:

a. Anti Sm antibody
b. Anti ds DNA antibody
c. Anti histoneantibody
d. Anti chromatin antibody

274. Cells involved in GVHD are?

a. Recipient B cells
b. Recipient T cells
c. Donor B cells
d. Donor T cells

275. After renal transplantation, which of the following is responsible for acute graft rejection?

a. C3a b. C3b
c. C5a d. C4d

276. For transplantation which HLA requires minimum matching

a. HLA A b. HLA B
c. HLA DR d. HLA DP

277. Which of the following is not a characteristic feature of GVHD?

a. Skin involvement
b. Renal involvement
c. Liver involvement
d. Intestinal involvement

278. Feature(s) of DiGeroge syndrome is/ are all except?

a. Results from failure of development of the third and fourth pharyngeal pouches
b. Absent thyroid
c. Absent parathyroid glands
d. B cell defect
e. Enhanced susceptibility of bacterial infection

266. a	267. c	268. b	269. b	270. a	271. d	272. c	273. a	274. d
275. d	276. d	277. b	278. d					

279. Chediak Higashi syndrome is characterised by the following except:

a. Neutrophilia
b. Defective degranulation
c. Delayed microbial killing
d. Giant granules

280. Which of the following are AIDS defining cancers? (PGI)

a. Burkitt's lymphoma
b. Non Hodgkin's Lymphoma
c. Ca esophagus
d. Primary lymphoma of brain
e. Invasive cancer of uterine cervix

281. Chemical nature of amyloid in hemodialysis associated amyloidosis

a. AA
b. AL
c. Aβ2
d. ATR

282. NK cells activity is enchanced by-

a. IL-1
b. TNF
c. IL-2
d. TGF-β

283. The primary function of toll-like reception is-

a. Vasodilation
b. Activation of immune system
c. Regulation of calcium channel
d. Second messenger

284. HLA-B27 is associated with-

a. Ankylosing spondylitis
b. Rheumatoid arthritis
c. Chronic active hepatitis
d. Diabetes

285. Type II hypersensitivity is mediated by (PGI type)

a. Immune complex
b. IgG
c. IgM
d. Complement
e. IgE

286. Which of the following immune hypersensitivity reaction is responsible for myasthenia gravis?

a. Type I hypersensitivity
b. Type II hypersensitivity
c. Type III hypersensitivity
d. Type IV hypersensitivity

287. Characteristic feature of SLE is-

a. Uveitis
b. Joint deformity
c. Polyserositis
d. Cavitating lesion in Lung

288. Drug induced lupus antibodies are found in-

a. Anti-Ro
b. Ds-DNA
c. Anti-Sm
d. Anti-histone antibody

289. Hemotoxylin bodies are seen in:

a. SLE
b. PAN
c. RA
d. Wegner's granulomatosis

290. Which type of amyloidosis is caused by mutation of the transthyretin protein?

a. Familial Mediterranean fever
b. Familial amyloidotic polyneuropathy
c. Dialysis associated amyloidosis
d. Prion protein associated amyloidosis

291. Major abnormalities in HIV include all of the following except

a. Increased IL-2
b. Loss of memory T cells
c. Decreased HLA II expression
d. Hypergammaglobulinemia

292. Th2 subset of T cells is induced by

a. Interferon gamma
b. IL-4
c. IL-17
d. IL-12

293. All of the following are examples of Type II hypersensitivity reactions except

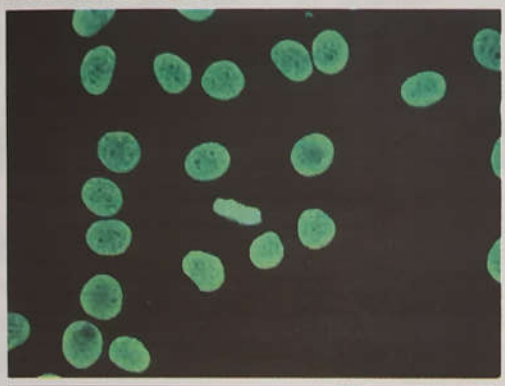
a. Autoimmune haemolytic anemia
b. Goodpasture syndrome
c. Acute rheumatic fever
d. Reactive arthritis

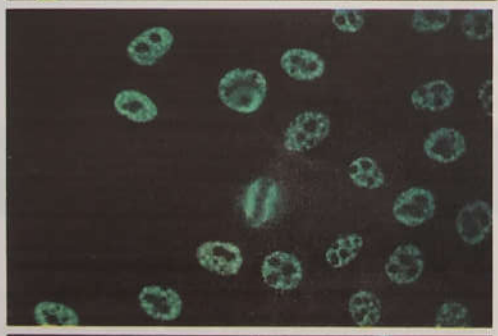
279. b	280. d,e	281. c	282. c	283. b	284. a	285. b,c,d	286. b	287. c
288. d	289. a	290. b	291. a	292. b	293. d			

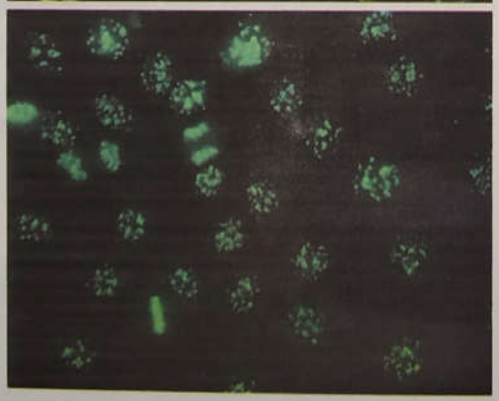
294. Most commonly implicated non MHC gene in autoimmunity is

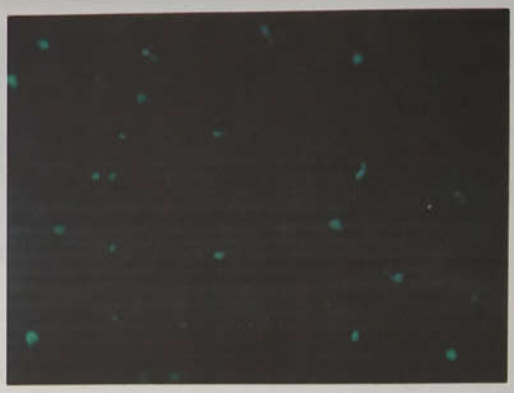
a. NOD2
b. Gene coding for CD 25
c. PTPN22
d. Gene coding for IL-7 receptor alpha

295. Drug induced SLE may show anti nuclear antibodies with the following staining pattern on immunofluorescence

a. 

b. 

c. 

d. 

296. Onion skin lesions in spleen are characteristically seen in

a. Systemic lupus erythematosus
b. Lymphoma infiltration
c. Hypersplenism
d. Sickle cell anemia

297. The earliest histologic finding in both the major and the minor salivary glands in Sjogren's syndrome is

a. Lymphoid follicles with germinal centres
b. Hyperplasia of ductal epithelial cells
c. Perivascular lymphoid infiltrate
d. All of the above

298. HLA associated with Multiple Sclerosis is

a. DRB1*0301 b. DQA1*0501
c. DRB1*1501 d. DQB1*1201

299. HLA matching is mandatory for all of the following organs except

a. Kidney b. Liver
c. Bone marrow d. Intestine

300. Leucocyte adhesion deficiency type I is caused by mutations in

a. CD 16 b. CD 17
c. CD 18 d. CD 117

294. c	295. a	296. a	297. c	298. c	299. b	300. c

Diseases of the Immune System – Explanations

266.A

IL-2 self activation

IFN for macrophages

267.C

Unusual nonmalignant lymphoproliferative disorder

Can be unicentric or multicentric

Most often involves lymph nodes but extranodal sites can also be affected

Three histological subtypes: hyaline vascular type, plasma cell type and mixed hyaline vascular plasma cell

Unicentric Castleman disease can affect all age groups and shows no predilection for either gender

Multicentric Castleman disease affects mainly HIV positive individuals; however, HIV negative individuals can also be affected

Most cases are KSHV / HHV8 associated

268.B

Class I MHC molecules are expressed on all nucleated cells and platelets and class II MHC molecules are mainly expressed on cells that present ingested antigens and respond to T-cell help (macrophages, B lymphocytes, and dendritic cells).

269.B

A protein called AIRE (autoimmune regulator) stimulates expression of some "peripheral tissue-restricted" self antigens in the thymus and is thus critical for deletion of immature T cells specific for these antigens. Mutations in the AIRE gene are the cause of an autoimmune polyendocrinopathy

270.A

Disease	Specificity of Autoantibody	% Positive	Association with Specific Disease Features
Systemic lupus erythematosus (SLE)	Double-stranded DNA	40-60	Nephritis; specific for SLE
	U1-RNP	30-40	Specific for SLE
	Smith (Sm) antigen (core protein of small RNP particles)	20-30	Congenital heart block; neonatal lupus
	Ro (SS-A)/La (SS-B) nucleoproteins	30-50	Antiphospholipid syndrome (in ~10% of SLE patients)
	Phospholipid-protein complexes (anti-PL)	30-40	
	Multiple nuclear antigens ("generic ANAs")	95-100	Found in other autoimmune diseases, not specific.
Systemic sclerosis	DNA topoisomerase 1	30-70	Diffuse skin disease, lung disease; specific for
	Centromeric proteins (CENPs) A, B, C	20-40	systemic sclerosis
	RNA polymerase III	15-20	Limited skin disease, ischemic digital loss, pulmonary hypertension
			Acute onset, scleroderma renal crisis, cancer
Sjogren syndrome	Ro/SS-A	70-95	
	La/SS-B		

Autoimmune myositis	Histidyl aminoacyl-tRNA synthetase, Jo1	25	Interstitial lung disease, Raynaud phenomenon
	Mi-2 nuclear antigen	5-10	Dermatomyositis, skin rash
	MDA5 (cytoplasmic receptor for viral RNA)	20-35 (Japanese)	Vascular skin lesions, interstitial lung disease
	TIF1 γ nuclear protein	15-20	Dermatomyositis, cancer
Rheumatoid arthritis	CCP (cyclic citrullinated peptides); various citrullinated proteins	60-80	Specific for rheumatoid arthritis
	Rheumatoid factor (not specific)	60-70	

271. D

Classification For Lupus Nephritis		
Class I	Minimal mesangial	Normal histology with mesangial deposits
Class II	Mesangial proliferation	Mesangial hypercellularity with expansion of the mesangial matrix
Class III	Focal nephritis	Focal endocapillary ± extracapillary proliferation with focal subendo-thelial immune deposits and mild mesangial expansion
Class IV	Diffuse nephritis	Diffuse endocapillary ± extracapillary proliferation with diffuse subendo-thelial immune deposits and mesangial alterations
Class V	Membranous nephritis	Thickened basement membranes with diffuse subepithelial immune deposits; may occur with class III or IV lesions and is sometimes called mixed membranous and proliferative nephritis
Class VI	Sclerotic nephritis	Global sclerosis of nearly all glomerular capillaries

272. C

Diffuse lupus nephritis (class IV) is the most common and severe form of lupus nephritis (Robbins)

273. A

- Homogeneous or diffuse nuclear staining usually reflects antibodies to chromatin, histones, and, occasionally, double-stranded DNA.
- Rim or peripheral staining patterns are most often indicative of antibodies to double-stranded DNA and sometimes to nuclear envelope proteins.
- Speckled pattern refers to the presence of uniform or variable-sized speckles. This is one of the most commonly observed patterns of fluorescence and therefore the least specific. It reflects the presence of antibodies to non-DNA nuclear constituents such as Sm antigen, ribonucleoprotein, and SS-A and SS-B reactive antigens.
- Nucleolar pattern refers to the presence of a few discrete spots of fluorescence within the nucleus and represents antibodies to RNA. This pattern is reported most often in patients with systemic sclerosis.
- Centromeric pattern. Patients with systemic sclerosis often contain antibodies specific for centromeres, which give rise to this pattern.

274. D

When immune-compromised recipients receive HSC preparations from allogeneic donors, the immunocompetent T cells present in the donor inoculum recognize the recipient's HLA antigens as foreign and react.

275. D

Acute antibody-mediated rejection is manifested mainly by damage to glomeruli and small blood vessels. Typically, the lesions consist of

inflammation of glomeruli and peritubular capillaries, associated with deposition of the complement breakdown product C4d, which is produced during activation of the complement system by the antibody dependent classical pathway

276. D

In kidney transplants, there is substantial benefit if all the polymorphic HLA alleles are matched (both inherited alleles of HLA-A, -B, and DR - being most important). However, HLA matching is usually not even done for transplants of liver, heart, and lungs, because other considerations, such as anatomic compatibility, severity of the underlying illness, and the need to minimize the time of organ storage, override the potential benefits of HLA matching.

277. B

Skin, liver and GIT are most commonly involved organs in GVHD

278. D

DiGeorge syndrome is a T-cell deficiency that results from failure of development of the third and fourth pharyngeal pouches. The latter give rise to the thymus, the parathyroids, some of the C cells of the thyroid, and the ultimobranchial body. Thus, individuals with this syndrome have a variable loss of T cell–mediated immunity (resulting from hypoplasia or lack of the thymus), tetany (resulting from lack of the parathyroids), and congenital defects of the heart and great vessels. In addition, the appearance of the mouth, ears, and facies may be abnormal. Absence of cell-mediated immunity is caused by low numbers of T lymphocytes in the blood and lymphoid tissues and poor defense against certain fungal and viral infections. The T-cell zones of lymphoid organs—paracortical areas of the lymph nodes and the periarteriolar sheaths of the spleen—are depleted. Ig levels may be normal or reduced, depending on the severity of the T-cell deficiency. In many cases, DiGeorge syndrome is not a familial disorder. It results from a deletion that maps to chromosome 22q11. This deletion is seen in more than 50% of patients, and DiGeorge syndrome is now considered a component of the 22q11 deletion syndrome, discussed in Chapter 5. One gene in the deleted region is TBX1, which is required for development of the branchial arch and the great vessels. Notably, TBX1 is involved by loss-of-function mutations in a few cases of DiGeorge syndrome that lack 22q11 deletions, strongly suggesting that its loss contributes to the observed phenotype.

279. B

Chédiak-Higashi syndrome is an autosomal recessive condition characterized by defective fusion of phagosomes and lysosomes, resulting in defective phagocytes function and susceptibility to infections. The main leukocyte abnormalities are neutropenia (decreased numbers of neutrophils), defective degranulation, and delayed microbial killing. Leukocytes contain giant granules, which can be readily seen in peripheral blood smears and are thought to result from aberrant phagolysosome fusion. In addition, there are abnormalities in melanocytes (leading to albinism), cells of the nervous system (associated with nerve defects), and platelets (causing bleeding disorders). The gene associated with this disorder encodes a large cytosolic protein called LYST, which is believed to regulate lysosomal trafficking.

280 D, E

Patients with AIDS have a high incidence of certain tumors, especially Kaposi sarcoma (KS), B-cell lymphoma, cervical cancer in women, and anal cancer in men. It is estimated that 25% to 40% of untreated HIV-infected individuals will eventually develop a malignancy. A common feature of these tumors is that they are caused by oncogenic DNA viruses, specifically Kaposi sarcoma herpesvirus (Kaposi sarcoma), EBV (B-cell lymphoma), and human papillomavirus (cervical and anal carcinoma). Even in healthy people, any of these viruses may establish latent infections that are kept in check by a competent immune system. The increased risk of malignancy in AIDS

patients exists mainly because of failure to contain the infections and reactivation of the viruses, as well as decreased immunity against the tumors.

NHL- most common malignancy in AIDS

281. C

Patients on longterm hemodialysis for renal failure can develop amyloidosis as a result of deposition of β2 -microglobulin. This protein is present in high concentrations in the serum of persons with renal disease and in the past, it was retained in the circulation because it could not be filtered through dialysis membranes. Patients sometimes presented with carpal tunnel syndrome because of β2 -microglobulin deposition. With new dialysis filters, the incidence of this complication has decreased substantially.

282. C

NK cells secrete cytokines such as interferon-γ (IFN-γ), which activates macrophages to destroy ingested microbes, and thus NK cells provide early defense against intracellular microbial infections. The activity of NK cells is regulated by many cytokines, including the interleukins IL-2, IL-15, and IL-12. IL-2 and IL-15 stimulate proliferation of NK cells, whereas IL-12 activates killing and secretion of IFN-γ.

283. B

The best known of the pattern recognition receptors are the Toll-like receptors (TLRs), whose founding member, Toll, was discovered in Drosophila. A family of related proteins was later shown to be essential for host defense against microbes. There are 10 TLRs in mammals, and each recognizes a different set of microbial molecules. The TLRs are present in the plasma membrane and endosomal vesicles . All these receptors signal by a common pathway that culminates in the activation of two sets of transcription factors: (1) NF-κB, which stimulates the synthesis and secretion of cytokines and the expression of adhesion molecules, both of which are critical for the recruitment and activation of leukocytes, and (2) interferon regulatory factors (IRFs), which stimulate the production of the antiviral cytokines, type I interferons

284. A

HLA Association - Robbins 9th

Condition	HLA Type
Sarcoidosis	Al, B8
Goodpasture's syndrome	DRB*1501, *1502
Celiac disease	DQ2, DQ8
Ankylosing spondylitis	627*2705, *2702
Reactive arthritis	B27
Primary sclerosing cholangitis	B8
Primary membranous nephropathy	DQA1
Grave's disease	DR3
Diabetes mellitusType 1	DR3, DR4, DQ8
Psoriasis	Cw*0602
Rheumatoid arthritis	DRB1
Psoriatic arthritis	B27 and Cw6
21 Hydroxylase deficiency	BW47
Hereditary hemochromatosis	A
Systemic lupus erythematosis	DRB1*0301, *1501
SLE with anti dsDNA, anti Sm and antiphospholipid antibodies	DQ
Subacute cutaneous SLE	DR3
Hydralazine induced SLE	DR4
Procainamide induced SLE	DR6
Sjogren's syndrome	B8, DR3, DRW52, DQA1/ B1
Behcet's disease	B51

285. B, C, D

Type	Immune Mechanisms	Histopathologic Lesions	Prototypical Disorders
Immediate (type I) hypersensitivity	Production of IgE antibody → immediate release of vasoactive amines and other mediators from mast cells; later recruitment of inflammatory cells	Vascular dilation, edema, smooth muscle contraction, mucus production, tissue injury, inflammation	Anaphylaxis; allergies; bronchial asthma (atopic forms)
Antibody-mediated (type II) hypersensitivity	Production of IgG, IgM → binds to antigen on target cell or tissue → phagocytosis or lysis of target cell by activated complement or Fc receptors; recruitment of leukocytes	Phagocytosis and lysis of cells; inflammation; in some diseases, functional derangements without cell or tissue injury	Autoimmune hemolytic anemia; Goodpasture syndrome
Immune complex-mediated (type III) hypersensitivity	Deposition of antigen-antibody complexes → complement activation recruitment → of leukocytes by complement products and Fc receptors → release of enzymes and other toxic molecules	Inflammation, necrotizing vasculitis (fibrinoid necrosis)	Systemic lupus erythematosus; some forms of glomerulonephritis; serum sickness; Arthus reaction
Cell-mediated (type IV) hypersensitivity	Activated T lymphocytes → (1) release of cytokines, inflammation and macrophage activation; (2) T cell-mediated cytotoxicity	Perivascular cellular infiltrates; edema; granuloma formation; cell destruction	Contact dermatitis; multiple sclerosis; type 1 diabetes; tuberculosis

286. B

Examples of type I hypersensitivity

Clinical Syndrome	Clinical and pathologic Manifestations
Anaphylaxis (may be caused by drugs, bee sting, food)	Fall in blood pressure (shock) cause by vascular dilation; airway obstruction due to laryngeal edema
Bronchial asthma	Airway obstruction caused by bronchial smooth muscle hyperactivity; inflammation and tissue injury caused by late-phase reaction
Allergic rhinitis, sinusitis (hay fever)	Increased mucus secretion; Inflammation of upper airways, sinuses
Food allergies	Increased peristalsis due to contraction of intestinal muscles

Examples of type II hypersensitivity

Disease	Target Antigen	Mechanisms of Disease
Autoimmune hemolytic anemia	Red cell membrane proteins (Mi blood group antigens. 1 antigen)	Opsonization and phagocytosis of red cells
Autoimmune thrombocytopenic purpura	Platelet membrane proteins (Gplla: illa integrin)	Opsonization and phagocytosis of platelets

Pemphigus vulgaris	Proteins In Intercellular Junctions of epidermal cells	Antibody-mediated activation of proteases, disruption of Intercellular adhesions
Vasculitis caused by ANCA	Neutrophil granule contents, presumably released from activated neutrophils	Neutrophil degeneration and Inflammation
Goodpasture syndrome	Noncollagenous protein In basement membranes of Kidney glomeruli and lung alveoli	Complement- and Fc receptor-mediated Inflammation
Acute rheumatic fever	Streptococcal cell wall antigen; antibody cross-reacts with myocardial antigen	Inflammation, macrophage activation
Myasthenia gravis	Acetylcholine receptor	Antibody Inhibits acetylcholine binding, down-modulates receptors
Graves disease (hyperthyroidism)	TSH receptor	Antibody-mediated stimulation of TSH receptors
Insult-resistant diabetes	Insulin receptor	Antibody inhibits binding of insulin
Pernicious anemia	Intrinsic factor of gastric parietal cells	Neutralization of Intrinsic factor, decreased absorption of vitamin B_{12}

Examples of type III hypersensitivity

Disease	Antigen Involved
Systemic lupus erythematosus	Nuclear antigens (circulating or "planted" In kidney)
Post streptococcal glomerulonephritis	Streptococcal cell wall antigen(s); maybe "planted" In glomerular basement membrane
Polyarteritis nodosa	Hepatitis B virus antigens In some cases
Reactive arthritis	Bacterial antigens (e.g., Yersinia)
Serum sickness	Various proteins, e.g., foreign serum protein (horse antithymocyte globulin!
Arthus reaction (experimental!	Various foreign proteins

Examples of type IV hypersensitivity

Disease	Specificity of Pathogenic T cells	Principal Mechanisms of tissue Injury
Rheumatoid arthritis	Collagen? Cirtullinated self-proteins?	Inflammation mediated by T_H 17 (and T_H1?) cytokines; role of antibiotics and immune complexes?
Multiple Sclerosis	Protein antigens in myelin (e.g., myelin basic protein)	Inflammation medicated by T_H1 and T_H17 cytokines, myelin destruction by activated macrophages
Type 1 diabetes Mellitus	Antigens of pancreatic islet ß cells (Insulin, glutamic acid decarboxylase, Other)	T cell-medicated inflammation, destruction of islet cells by CTLs
Inflammatory bowel diseases	Enteric bacteria: self-antigens?	Inflammation mediated by T_H1 and T_H17 cytokines
Psoriasis	Unknown	Inflammation mediated mainly by T_H17 cytokines
Contact sensitivity	Various environmental chemicals (e.g., urushiol from poison ivy or poison oak)	Inflammation mediated by T_H1(and T_H17?) cytokines

287. C

SLICC Classification Criteria for Systemic Lupus Erythematosus

Requirements: ≥4 criteria (at least 1 clinical and 1 laboratory criteria) OR biopsy-proven lupus nephritis with positive ANA or Anti-DNA

Clinical Criteria	Immunologic Criteria
1. Acute Cutaneous Lupus*	1. ANA
2. Chronic Cutaneous Lupus*	2. Anti-DNA
3. Oral or nasal ulcers *	3. Anti-Sm
4. Non-scarring alopecia	4. Antiphospholipid Ab*
5. Arthritis * 5. Low complement (C3, C4, CH50)	
6. Serositis *	6. Direct Coombs' test (do not count in the presence of hemolytic anemia)
7. Renal *	
8. Neurologic *	
9. Hemolytic anemia	
10. Leukopenia *	
11.Thrombocytopenia (<100,000/mm3)	

288. D

A lupus erythematosus-like syndrome may develop in patients receiving a variety of drugs, including hydralazine, procainamide, isoniazid, and D-penicillamine, to name only a few. Somewhat surprisingly, anti-TNF therapy, which is effective in rheumatoid arthritis and other autoimmune diseases, can also cause drug-induced lupus. Many of these drugs are associated with the development of ANAs, but most patients do not have symptoms of lupus erythematosus. For example, 80% of patients receiving procainamide test positive for ANAs, but only one third of these manifest clinical symptoms, such as arthralgias, fever, and serositis. Although multiple organs are affected, renal and central nervous system involvement is distinctly uncommon. There are serologic and genetic differences from classic SLE, as well. Antibodies specific for double-stranded DNA are rare, but there is an extremely high frequency of antibodies specific for histones. Persons with the HLA-DR4 allele are at a greater risk of developing a lupus erythematosus-like syndrome after administration of hydralazine, whereas those with HLA-DR6 (but not DR4) are at high risk with procainamide. The disease remits after withdrawal of the offending drug.

289. A

There is no evidence that ANAs, which are involved in immune complex formation, can penetrate intact cells. If cell nuclei are exposed, however, the ANAs can bind to them. In tissues, nuclei of damaged cells react with ANAs, lose their chromatin pattern, and become homogeneous, to produce so-called LE bodies or hematoxylin bodies. Related to this phenomenon is the LE cell, which is readily seen when blood is agitated in vitro. The LE cell is any phagocytic leukocyte (blood neutrophil or macrophage) that has engulfed the denatured nucleus of an injured cell. The demonstration of LE cells in vitro was used in the past as a test for SLE. With new techniques for detection of ANAs, however, this test is now largely of historical interest. Sometimes, LE cells are found in pericardial or pleural effusions in patients.

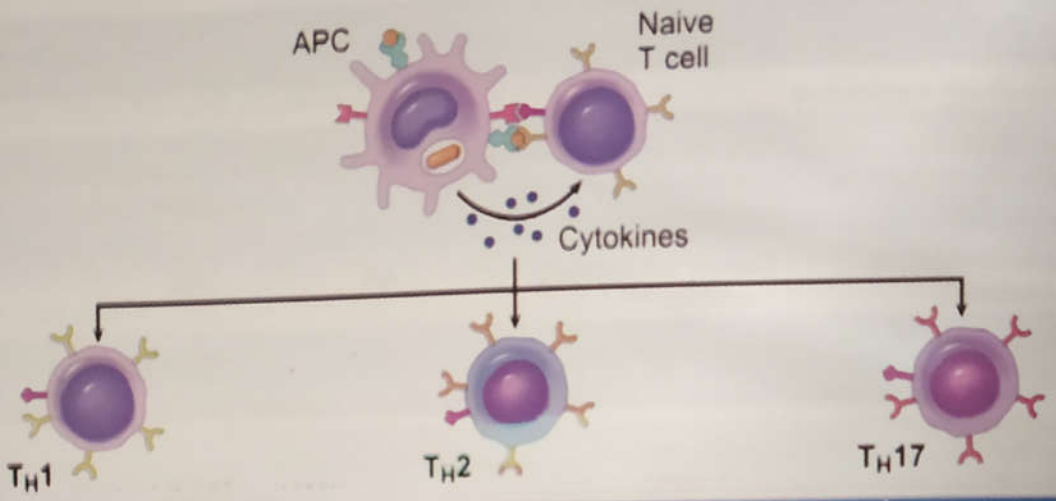
290. B

Clinicopathologic Category	Associated Diseases	Major Fibril Protein	Chemically Related Precursor Protein
Systemic (Generalized) Amyloidosis			
Immunocyte dyscrasias with amyloidosis (primary amyloidosis)	Multiple myeloma and other monoclonal plasma cell proliferations	AL	Immunoglobulin light chains, chiefly λ type
Reactive systemic amyloidosis (secondary amyloidosis)	Chronic inflammatory conditions	AA	SAA
Hemodialysis-associated amyloidosis	Chronic renal failure	Aβ2m	β2-microglobulin
Hereditary Amyloidosis			
Familial Mediterranean fever		AA	SAA
Familial amyloidotic neuropathies (several types)		ATTR	Transthyretin
Systemic Senile Amyloidosis		ATTR	Transthyretin
Localized Amyloidosis			
Senile cerebral	Alzheimer disease	Ab	APP
Endocrine		A Cal	Calcitonin
Medullary carcinoma of thyroid	Type 2 diabetes	AIAPP	Islet amyloid peptide
Islets of Langerhans		AANF	Atrial natriuretic factor
Isolated atrial amyloidosis			

291. A

Lymphopenia
Predominantly caused by selective loss of the CD4+ helper T-cell subset
Decreased T-Cell Function In Vivo
Preferential loss of activated and memory T cells Decreased delayed-type hypersensitivity Susceptibility to opportunistic infections Susceptibility to neoplasms
Altered T-Cell Function In Vitro
Decreased proliferative response to mitogens, alloantigens, and soluble antigens Decreased cytotoxicity Decreased helper function for B-cell antibody production Decreased IL-2 and IFN-γ production
Polyclonal B-Cell Activation
Hypergammaglobulinemia and circulating immune complexes Inability to mount de novo antibody response to new antigens Poor responses to normal B-cell activation signals in vitro
Altered Monocyte or Macrophage Functions
Decreased chemotaxis and phagocytosis Decreased class II HLA expression Diminished capacity to present antigen to T cells

292. B



Major cytokines produced	IFN-γ	IL-4, IL-5, IL-13	IL-17, IL-22
Cytokines that induce this subset	IFN-γ, IL-12	IL-4	TGF-β, IL-6, IL-1, IL-23
Immunological reactions triggered	Macrophage activation, stimulation of IgG antibody production	Stimulation of IgE production, activation of mast cells and eosinophils	Recruitment of neutrophils, monocytes
Host defense against	Intracellular microbes	Helminthic parasites	Extracellular bacteria, fungi
Role in disease	Autoimmune and other chronic inflammatory diseases (such as IBD, psoriasis, granulomatous inflammation)	Allergies	Autoimmune and other chronic inflammatory diseases (such as IBD, psoriasis, MS)

293. D

Disease	Target Antigen	Mechanisms of Disease	Clinicopathologic Manifestations
Autoimmune hemolytic anemia	Red cell membrane proteins (Rh blood group antigens, I antigen)	Opsonization and phagocytosis of red cells	Hemolysis, anemia
Autoimmune thrombocytopenic purpura	Platelet membrane proteins (GpIIb:IIIa integrin)	Opsonization and phagocytosis of platelets	Bleeding
Pemphigus vulgaris	Proteins in intercellular junctions of epidermal cells (epidermal cadherin)	Antibody-mediated activation of proteases, disruption of intercellular adhesions	Skin vesicles (bullae)
Vasculitis caused by ANCA	Neutrophil granule proteins, presumably released from activated neutrophils	Neutrophil degranulation and inflammation	Vasculitis
Goodpasture syndrome	Noncollagenous protein in basement membranes of kidney glomeruli and lung alveoli	Complement- and Fc receptor-mediated inflammation	Nephritis, lung hemorrhage

Acute rheumatic fever	Streptococcal cell wall antigen; antibody cross-reacts with myocardial antigen	Inflammation, macrophage activation	Myocarditis, arthritis
Myasthenia gravis	Acetylcholine receptor	Antibody inhibits acetylcholine binding, down-modulates receptors	Muscle weakness, paralysis
Graves disease (hyperthyroidism)	TSH receptor	Antibody-mediated stimulation of TSH receptors	Hyperthyroidism
Insulin-resistant diabetes	Insulin receptor	Antibody inhibits binding of insulin	Hyperglycemia, ketoacidosis
Pernicious anemia	Intrinsic factor of gastric parietal cells	Neutralization of intrinsic factor, decreased absorption of vitamin B1?	Abnormal erythropoiesis, anemia

294. C

Polymorphisms in a gene called PTPN22, which encodes a protein tyrosine phosphatase, are associated with rheumatoid arthritis, type 1 diabetes, and several other autoimmune diseases. Because these disorders have a fairly high prevalence (especially rheumatoid arthritis), PTPN22 is said to be the gene that is most frequently implicated in autoimmunity. It is postulated that the disease-associated variants encode a phosphatase that is functionally defective and is thus unable to fully control the activity of tyrosine kinases, which are involved in many responses of lymphocytes and other cells. The net result is excessive lymphocyte activation.

295. A

Drug induced SLE shows anti histone antibodies which have a homogenous staining.

- Homogeneous or diffuse nuclear staining usually reflects antibodies to chromatin, histones, and, occasionally, double-stranded DNA.
- Speckled pattern refers to the presence of uniform or variable-sized speckles. This is one of the most commonly observed patterns of fluorescence and therefore the least specific. It reflects the presence of antibodies to non-DNA nuclear constituents such as Sm antigen, ribonucleoprotein, and SS-A and SS-B reactive antigens.
- Nucleolar pattern refers to the presence of a few discrete spots of fluorescence within the nucleus and represents antibodies to RNA. This pattern is reported most often in patients with systemic sclerosis.
- Centromeric pattern. Patients with systemic sclerosis often contain antibodies specific for centromeres, which give rise to this pattern.

296. A

Central penicilliary arteries may show concentric intimal and smooth muscle cell hyperplasia, producing so-called onion-skin lesions.

297. C

Lacrimal and salivary glands are the
major targets of the disease, although other exocrine glands,
including those lining the respiratory and gastrointestinal tracts
and the vagina, may also be involved. The earliest histologic
finding in both the major and the minor salivary glands is periductal
and perivascular lymphocytic infiltration. Eventually the
lymphocytic infiltrate becomes extensive, and in the larger salivary glands lymphoid follicles with germinal centers
may be seen. The ductal lining epithelial cells may show hyperplasia,
thus obstructing the ducts. Later there is atrophy of the

acini, fibrosis, and hyalinization; still later in the course atrophy and replacement of parenchyma with fat are seen.

298. C

Disease	HLA allele
Rheumatoid arthritis (anti-CCP Ab positive)	DRB1,1 SE allele[1] DRB1, 2 SE alleles
Type 1 diabetes	DRB1 *0301 -DQA1*0501 - DQB1*0201 haplotype DRB1*0401-DQA1*0301-DQB1*0302 haplotype DRB1*0301/0401 haplotype heterozygotes
Multiple sclerosis	DRB1 *1501
Systemic lupus erythematosus	DRB1 *0301 DRB1*1501
Ankylosing spondylitis	B*27 (mainly B*2705 and B*2702)
Celiac disease	DQA1 *0501 -DQB1*0201 haplotype

299. B

The value of HLA matching between donor and recipient varies in different solid-organ transplants. In kidney transplants, there is substantial benefit if all the polymorphic HLA alleles are matched (both inherited alleles of HLA-A, -B, and DR). However, HLA matching is usually not even done for transplants of liver, heart, and lungs, because other considerations, such as anatomic compatibility, severity of the underlying illness, and the need to minimize the time of organ storage, override the potential benefits of HLA matching.

300. C

Disease	Defects
Defects in Leukocyte Function	
Leukocyte adhesion deficiency 1	Defective leukocyte adhesion because of mutations in β chain of CD11/CD18 integrins
Leukocyte adhesion deficiency 2	Defective leukocyte adhesion because of mutations in fucosyl transferase required for synthesis of sialylated oligosaccharide (receptor for selectins)
Chediak-Higashi syndrome	Decreased leukocyte functions because of mutations affecting protein involved in lysosomal membrane traffic
Chronic granulomatous disease	Decreased oxidative burst
X-linked	Phagocyte oxidase (membrane component)
Autosomal recessive	Phagocyte oxidase (cytoplasmic components)
Myeloperoxidase deficiency	Decreased microbial killing because of defective MP0-H2O2 system
Defects in the Complement System	
C2, C4 deficiency	Defective classical pathway activation, results in reduced resistance to infection and reduced clearance of immune complexes
C3 deficiency	Defects in all complement functions
Deficiency of complement regulatory proteins	Excessive complement activation; clinical syndromes include angioedema, paroxysmal hemoglobinuria, others

Neoplasia – Questions

301. Lack of differentiation is called?

a. Anaplasia b. Metaplasia
c. Dysplasia d. Carcinoma

302. C-KIT mutations are seen in-

a. Gastrointestinal stromal tumors
b. Ovarian cancers
c. Neuroblastoma
d. Small-cell carcinoma of lung

303. Number of cancer cells present in the smallest clinically detectable mass-

a. 10^3 cell b. 10^6 cell
c. 10^9 cell d. 10^{12} cell

304. Overgrowth of a bile duct at localized region is?

a. Hamartoma
b. Malignant tumor
c. Choriostoma
d. Polyp

305. Which of the following mutation is seen in Cowden syndrome?

a. PTEN mutation
b. STK11 mutation
c. PTCH mutation
d. SMAD4 mutation

306. Which of the following Dyads are correctly matched? (PGI)

a. RB1-retinoblastoma
b. PTEN-melanoma
c. BRCA2-Breast cancer
d. n-MYC-Neuroblastoma
e. WT1=Wilm's tumor

307. If the RB gene phosphorylation is defective which of the following will happen?

a. Cell cycle will stop at G2
b. Cell cycle will stop at G1
c. There will be no effect on cell cycle as RB gene phosphorylation is not needed
d. The cell cycle progresses and cell divides

308. E6 protein of high risk human papilloma viruses bind to the following protein and there by promotes carcinogenesis:

a. RB b. TP53
c. APC d. MDM2

309. All are associated with BRCA mutation except:

a. Ovarian carcinoma
b. Prostate carcinoma
c. Endometrial carcinoma
d. Papillary serous cancer of peritoneum

310. BRCA 1 and BRCA 2 genes are located on chromosomes:

a. 13 and 17 b. 17 and 22
c. 17 and 13 d. 13 and 22

311. Gene promoting metastasis:

a. SNAIL b. TWIST
c. Both d. None

312. Excessive fibrosis in a tumor in called:

a. Anaplasia b. Metaplasia
c. Desmoplasia d. Dysplasia

313. Cancers associated with viruses? (PGI)

a. Hepatocellular cancer
b. Kaposi sarcoma
c. Nasopharyngeal cancer
d. Small cell Ca lung
e. Prostatic Ca

301. a	302. a	303. c	304. a	305. a	306. a,c,d,e	307. d	308. a	309. c
310. c	311. c	312. c	313. a,b,e					

314. Chromopthysis mechanism of carcinogenesis has been found to be associated with which of the following tumors?

a. Sarcomas
b. Osteosarcoma
c. RCC
d. All

315. Paraneoplastic syndrome(s) associated with lymphoma?

a. SIADH
b. Hypercalcemia
c. Cushing's syndrome
d. Acanthosis nigricans

316. In tumor lysis syndrome, all of the following are seen except:

a. Hyperuricemia
b. Hypercalcemia
c. Hyperkalemia
d. Hyperphosphatemia

317. In CA breast, based on Which stage/ grade of IHC staining, FISH for gene amplification will be done?

a. Her2 neu 3+
b. Her2 neu 2+
c. Her2 neu 1+
d. Will be done irrespective of above

318. A surgeon suspecting testicular carcinoma in a patient asks the intern to send the sample for histopathology, what is the fluid in which the intern should send the sample to the pathologist?

a. Bouin solution
b. 10% formalin
c. 95% ethanol
d. Alcohol

319. Neuroendocrine cell tumor markers are: (PGI)

a. Chromogranin A
b. CD56
c. Neuron-specific enolase
d. Synaptophysin
e. Cytokeratin 7

320. Tumor markers and tumors-find the wrong match:

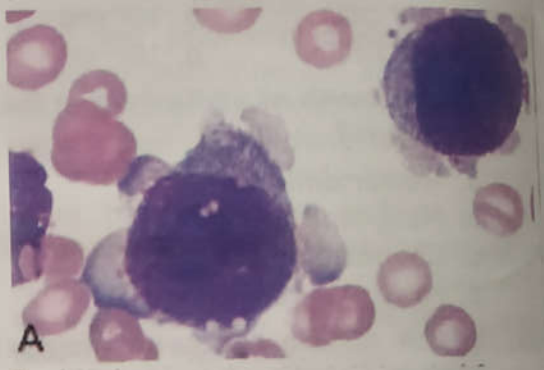
a. Desmin-Carcinoma
b. Vimentin-sarcoma
c. Leucocyte specific antigen-lymphoma
d. S100-melanoma

321. All of the following viruses have been implicated in causation of hematolymphoid neoplasms except

a. EBV
b. CMV
c. HHV8
d. HTLV

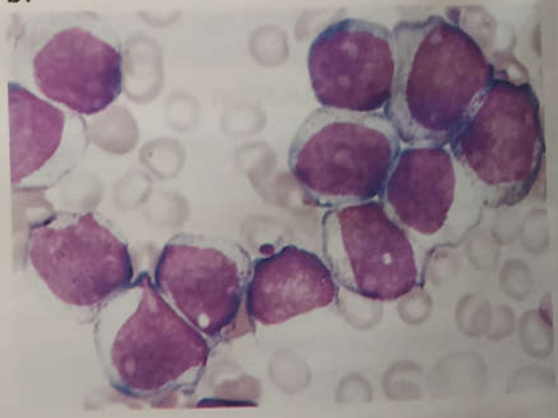
322. A child with Down's syndrome presented with increased TLC and anemia. On performing differential count, 65% atypical cells were seen. The diagnosis of AML M7 was rendered. The following morphology in addition to flow cytometry helped in the identification of the condition

a.

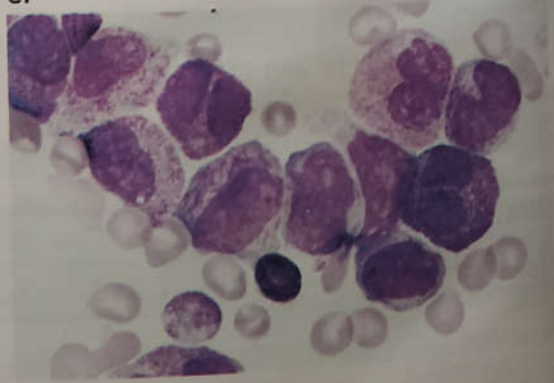


A

b.

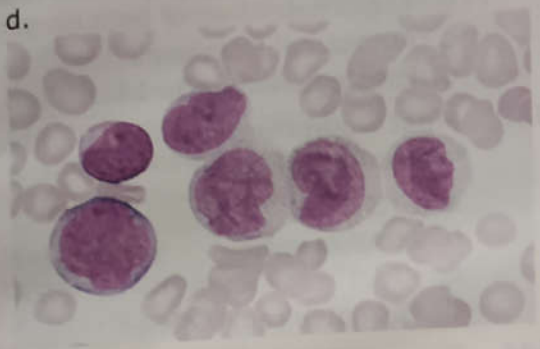


c.



314. b	315. b	316. b	317. b	318. a	319. a,b,c,d	320. a	321. b	322. a

d.



323. A 45-year man presents a cervical lymphadenopathy, biopsy from the lesion closely adhesive neoplastic cells forming glands. A suspicion of metastatic adenocarcinoma was made. Further imaging and investigations does not reveal any possible primary. A further Immunohistochemistry shows CK7+, CK20- . which of the following organs is most likely organ for such a primay

a. Lung
b. Liver
c. Colon
d. Prostate

324. Which of the following cyclins propels cell from G2 --> M phase

a. Cyclin B
b. Cyclin D
c. Cyclin A
d. Cyclin E

325. Philadephia chromosome seen in chronic myeloid leukemia produces a protein called

a. P170
b. P210
c. P190
d. P230

326. Loss of which of the following protein is implicated in aiding metastasis of tumor cells?

a. Integrins
b. Cadherins
c. Matrix metalloproteases
d. Cathepsin

327. Ductal carcinoma in situ can be differentiated from invasive ductal carcinoma by all of the following except

a. Nuclear pleomorphism
b. Basement membrane invasion
c. Loss of myoepithelial cells
d. Central areas of necrosis

328. All of the following points are true about Lynch syndrome except

a. Left colon is involved more
b. Endometrial cancer is the most common extra colonic tumor involved
c. Intra tumoral lymphocytes are more
d. Seen in younger age group

329. Which of the following is the most reliable feature of malignancy in a paraganglioma?

a. Presence of metastasis
b. Nuclear pleomorphism
c. Vascular or capsular invasion
d. Increased mitotic activity

330. Which of the following is not true regarding genetics of thyroid tumors

a. Driver mutations in KRAS are seen in follicular carcinoma
b. BRAF mutations are the most common mutations seen in papillary carcinoma
c. RET rearrangements are seen in medullary carcinoma
d. PI3K mutations are seen in anaplastic carcinoma

331. All are markers for malignant melanoma except

a. HMB 45
b. MART1
c. SOX 11
d. SOX 10

332. All of the following familial cutaneous cancer syndromes show autosomal dominant inheritance except

a. Muir Torre syndrome
b. Cowden syndrome
c. Gorlin syndrome
d. Ataxia telangiectasia

333. All are true about aflatoxin except

a. Causes hepatocellular carcinoma
b. Aflatoxin B3 mostly implicated
c. Causes P53 mutation in codon 249
d. Arginine to serine substitution

334. Which of the following Is the most implicated in causation of cutaneous cancers?

a. UVA
b. UVB
c. UVC
d. All of the above

323. a	324. a	325. b	326. b	327. a	328. a	329. a	330. c	331. c
332. d	333. b	334. b						

335. Epigenetic changes in ARID1A is seen in

a. AML
b. Follicular lymphoma
c. Ovarian clear cell carcinoma
d. Renal cell carcinoma

336. Which of the following gene is translocated in Burkitt lymphoma?

a. PML
b. FLI1
c. MYC
d. CCND1

337. Upregulation of which of the following has been noted on tumor cells which helps them evade immune surveillance

a. PD L1
b. CD 28
c. CTLA4
d. CD 86

338. Which IHC marker will be useful to distinguish between ALL and thymoma

a. TDT
b. CD3
c. CD1a
d. Cytokeratin

339. Which of the following is the most common malignant tumor in adult males in India?

a. Lung cancer
b. Oropharyngeal carcinoma
c. Gastric carcinoma
d. Colorectal carcinoma

340. Which is not a tumor suppressor gene? (PGI)

a. P53
b. CD95
c. RAS
d. PTEN
e. Stk 7/Stk11

341. On which cell cycle checkpoints BRCA-2 acts?

a. G2-M
b. G1-M
c. M phase
d. G1 phase
e. S phase

342. All are true regarding Retinoblastoma except?

a. Play major role in cell cycle regulation
b. Require deletion of both Rb genes
c. Autosomal Dominant
d. Located on chr 13p 14

343. Most common gene involved in endometrial ca is?

a. PTEN
b. BRAF mutation
c. KRAS
d. Mismatch repair genes

344. The following protein is called "Gatekeeper of colonic neoplasia":

a. APC
b. STK11
c. SMAD2
d. PTEN

345. Hall mark mechanism of tumor suppressor gene inactivation-

a. Loss of heterozygosity
b. DNA hypermethylation
c. DNA hypomethylation
d. Histone deacetylation

346. CDK4 positive tumors are? (PGI)

a. Melanomas
b. Sarcomas
c. Glioblastomas
d. Lobular Ca breast
e. Prostate Ca

347. Dysplasia is characterized by all except:

a. High nuclear to cytoplasmic ratio
b. Loss of architecture
c. Pleomorphism
d. Invasion

348. Which is the most common site of distant metastasis:

a. Lung
b. Bone
c. Liver
d. Brain

349. All of the following are involve in tumor metastasis cascade except:

a. Fibronectin
b. E-cadherin
c. Type IV collagenase
d. Tyrosine kinase

335. c	336. c	337. a	338. d	339. a	340. c	341. d	342. d	343. a
344. a	345. a	346. a,b,c	347. d	348. a	349. d			

350. Human papilloma virus is/are associated with all except: (PGI)
a. Oropharyngeal tumors
b. Carcinoma nasopharynx
c. Carcinoma anal canal
d. Carcinoma pancreas
e. Carcinoma cervix

351. Malignancy associated with hypercalcemia:
a. Breast cancer
b. Prostate cancer
c. Small cell lung cancer
d. Non small lung cancer

352. Migratory thrombophlebitis is seen in all except?
a. Lung Ca
b. Prostate Ca
c. Colon Ca
d. Pancreatic Ca

353. Which of the following immunohistochemical marker is positive in neuroendocrine tumor?
a. Cytokeratin
b. Synaptophysin
c. Calretinin
d. GFAP

354. Which of the following neoplasms shows ALK positivity?
a. Ewing sarcoma
b. Inflammatory myofibroblastic tumor
c. Synovial sarcoma
d. Fibromatosis

355. Fixative agent for PAP smear?
a. Norma saline
b. Formalin
c. 95% ethanol
d. Air drying

356. ALK1 mutation seen mc with which hereditary hemorrhagic telangiectasia?
a. Type 1
b. Type 2
c. Type 3
d. Type 4

357. Carbohydrate marker of recurrent breast cancer is?
a. CA 15.3
b. CA 125
c. CEA
d. CA 19.9

358. For detection of carcinoma lip, stain used is?
a. Giemsa
b. Crystal violet
c. Toulidine blue
d. Hematoxylin and eosin

359. AFP and CEA both rise in:
a. Testicular tumors
b. Hepatocellular carcinoma
c. Renal cell carcinoma
d. Germ cell tumor of ovary

360. Grading of cancers does not depend on:
a. Degree of differentiation
b. Number of mitoses
c. Architectural features
d. Size of primary lesion

361. HMB-45 is positive in:
a. Small cell carcinoma of lung
b. Adenocarcinoma of lung
c. Squamous cell carcinoma of lung
d. Angiomyolipoma of lung

362. An undifferentiated malignant tumor on immuno-histochemical stain shows cytoplasmic positively of most of the tumor cells for cytokeratin. The most probable diagnosis of the tumor is:
a. Lymphoma
b. Carcinoma
c. Sarcoma
d. Malignant melanoma

363. Which of the following is not premalignant?
a. Hyperplasia
b. Metaplasia
c. Hypertrophy
d. Dysplasia

350. d	351. a	352. a	353. b	354. b	355. c	356. b	357. a	358. c	359. b
360. d	361. d	362. b	363. c						

Neoplasia – Explanations

301.A

Tumors composed of undifferentiated cells are said to be anaplastic, a feature that is a reliable indicator of malignancy. The term anaplasia literally means "backward formation"—implying dedifferentiation, or loss of the structural and functional differentiation of normal cells

302.A

Growth Factors			
PDGF-p chain	PDGFB	Overexpression	Astrocytoma
Fibroblast growth factors	HST1 FGF3	Overexpression Amplification	Osteosarcoma Stomach cancer Bladder cancer Breast cancer Melanoma
TGF-α	TGFA	Overexpression	Astrocytomas
HGF	HGF	Overexpression	Hepatocellular carcinomas Thyroid cancer
Growth Factor Receptors			
EGF-receptor family	ERBB1 (EGFR) ERBB2 (HER)	Mutation Amplification	Adenocarcinoma of lung Breast carcinoma
FMS-like tyrosine kinase 3	FLT3	Point mutation	Leukemia
Receptor for neurotrophic factors	RET	Point mutation	Multiple endocrine neoplasia 2A and B, familial medullary thyroid carcinomas
PDGF receptor	PDGFRB	Overexpression, translocation	Gliomas, leukemias
Receptor for KIT ligand	KIT	Point mutation	Gastrointestinal stromal tumors, seminomas, leukemias
ALK receptor	ALK	Translocation, fusion gene formation Point mutation	Adenocarcinoma of lung, certain lymphomas Neuroblastoma
Proteins Involved in Signal Transduction			
GTP-binding (G) proteins	KRAS HRAS NRAS GHAO GAMS	Point mutation Point mutation Point mutation Point mutation Point mutation	Colon, lung, and pancreatic tumors Bladder and kidney tumors Melanomas, hematologic malignancies Uveal melanoma Pituitary adenoma, other endocrine tumors

Nonreceptor tyrosine kinase	ABL	Translocation Point mutation	Chronic myelogenous leukemia Acute lymphoblastic leukemia
RAS signal transduction	BRAF	Point mutation, Translocation	Melanomas, leukemias, colon carcinoma, others
Notch signal transduction	NOTCH!	Point mutation, Translocation Gene rearrangement	Leukemias, lymphomas, breast carcinoma
JAK/STAT signal transduction	JAK2	Translocation	Myeloproliferative disorders Acute lymphoblastic leukemia
Nuclear Regulatory Proteins			
Transcriptional activators	MYC NMYC	Translocation Amplification	Burkitt lymphoma Neuroblastoma
Cell Cycle Regulators			
Cyclins	CCND1 (Cyclin D1)	Translocation Amplification	Mantle cell lymphoma, multiple myeloma Breast and esophageal cancers
Cyclin-dependent kinase	CDK4	Amplification or point mutation	Glioblastoma, melanoma, sarcoma

303.C

- Original untransformed cell- 10 µm
- 30 population doublings to produce 10^9 cells- 1 gm- smallest clinically detectable mass (*)
- 10 additional doubling cycles to produce 10^{12} cells- approximately 1 kg – maximal size compatible with life

304.A

Hamartoma is a mass of disorganized tissue indigenous to the particular site, such as the lung or the liver.

While traditionally considered developmental malformations, many hamartomas have clonal chromosomal aberrations that are acquired through somatic mutations and on this basis are now considered to be neoplastic

3005. A

PTEN:

- Encodes a lipid phosphatase which is an important negative regulator of PI3K/AKT pathway
- Germline LoF mutations in Cowden syndrome

PTCH1

- Encodes membrane receptor that is a negative regulator of Hedgehog signalling pathway
- Germline LoF cause Gorlin syndrome associated with high risk of basal cell carcinoma and medulloblastoma

STK11

- Serine threonine kinase 11
- Peutz Jegher syndrome

SMAD4

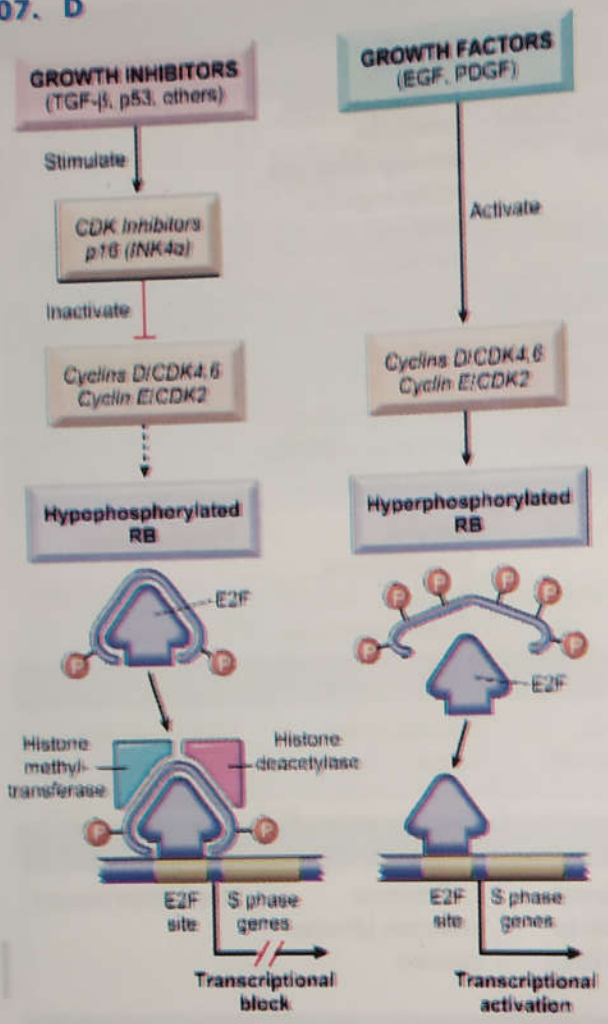
- Signal transduction protein of TGF beta pathway
- Pancreatic cancer
- Juvenile polyposis

306.A, C, D, E

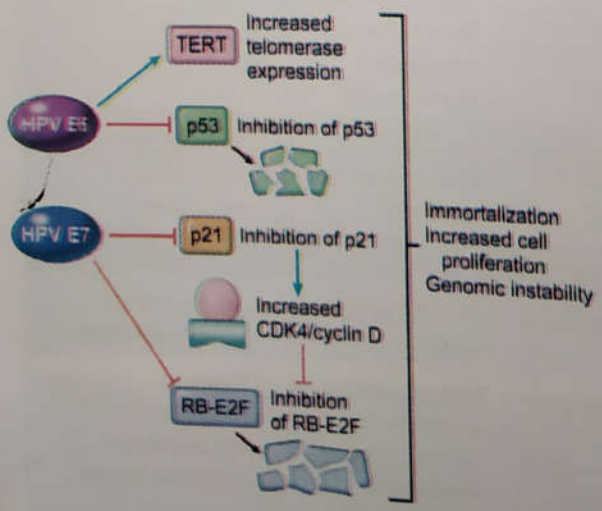
Inhibitors of Mitogenic Signaling Pathways				
APC	Adenomatous polyposis coli protein	Inhibitor of WNT signaling	Familial colonic polyps and carcinomas	Carcinomas of stomach, colon, pancreas; melanoma
NF1	Neurofibromin-1	Inhibitor of RAS/MAPK signaling	Neurofibromatosis type 1 (neurofibromas and malignant peripheral nerve sheath tumors)	Neuroblastoma, juvenile myeloid leukemia
NF2	Merlin	Cytoskeletal stability, Hippo pathway signaling	Neurofibromatosis type 2 (acoustic schwannoma and meningioma)	Schwannoma, meningioma
PTCH	Patched	Inhibitor of Hedgehog signaling	Gorlin syndrome (basal cell carcinoma, medulloblastoma, several benign tumors)	Basal cell carcinoma, medulloblastoma
PTEN	Phosphatase and tensin homologue	Inhibitor of PI3K/AKT signaling	Cowden syndrome (variety of benign skin, GI, and CNS growths; breast, endometrial, and thyroid carcinoma)	Diverse cancers, particularly carcinomas and lymphoid tumors
SMAD2, SMAD4	SMAD2, SMAD4	Component of the TGFβ signaling pathway, repressors of MYC and CDK4 expression, inducers of CDK inhibitor expression	Juvenile polyposis	Frequently mutated (along with other components of the TGFJ3 signaling pathway) in colonic and pancreatic carcinoma
Inhibitors of Cell Cycle Progression				
RB	Retinoblastoma (RB) protein	Inhibitor of G,/S transition during cell cycle progression	Familial retinoblastoma syndrome (retinoblastoma, osteosarcoma, other sarcomas)	Retinoblastoma; osteosarcoma carcinomas of breast, colon, lung
CDKN2A	p16/INK4a and p14/ARF	p16: Negative regulator of cyclin-dependent kinases; p14, indirect activator of p53	Familial melanoma	Pancreatic, breast, and esophageal carcinoma, melanoma, certain leukemias

Inhibitors of "Pro-growth" Programs of Metabolism and Angiogenesis				
VHL	Von Hippel Lindau (VHL) protein	Inhibitor of hypoxia-induced transcription factors (e.g., HIFIa)	Von Hippel Lindau syndrome (cerebellar hemangioblastoma, retinal angioma, renal cell carcinoma)	Renal cell carcinoma
STK11	Liver kinase B1 (LKB1) or STK11	Activator of AMPK family of kinases; suppresses cell growth when cell nutrient and energy levels are low	Peutz-Jeghers syndrome (GI polyps, GI cancers, pancreatic carcinoma and other carcinomas)	Diverse carcinomas (5%-20% of cases, depending on type)
SDHB, SDHD	Succinate dehydrogenase complex subunits B and D	TCA cycle, oxidative phosphorylation	Familial paraganglioma, familial pheochromocytoma	Paraganglioma
Inhibitors of Invasion and Metastasis				
CDHl	E-cadherin	Cell adhesion, inhibition of cell motility	Familial gastric cancer	Gastric carcinoma, lobular breast carcinoma
Enablers of Genomic Stability				
TP53	p53 protein	Cell cycle arrest and apoptosis in response to DNA damage	Li-Fraumeni syndrome (diverse cancers)	Most human cancers
DNA Repair Factors				
BRCA1, BRCA2	Breast cancer-1 and breast cancer-2 (BRCA1 and BRCA2)	Repair of double-stranded breaks in DNA	Familial breast and ovarian carcinoma; carcinomas of male breast; chronic lymphocytic leukemia (BRCA2)	Rare f
MSH2, MLH1, MSH6	MSH1, MLH1, MSH6	DNA mismatch repair	Hereditary nonpolyposis colon carcinoma	Colonic and endometrial carcinoma
Unknown Mechanisms				
WT1	Wilms tumor-1 (WT1)	Transcription factor	Familial Wilms tumor	Wilms tumor, certain leukemias
MEN1	Menin	Transcription factor	Multiple endocrine neoplasia-1 (MEN1; pituitary, parathyroid, and pancreatic endocrine tumors)	Pituitary, parathyroid, and pancreatic endocrine tumors

307. D



308. A



309. C

Although the precise causes are not known, a link with certain variants of BRCA1/2 has been described. [3] Furthermore, women with BRCA1/2 mutation have a 5% risk of developing primary peritoneal cancer even after prophylactic oophorectomy.

Primary peritoneal carcinoma shows similar rates of tumor suppressor gene dysfunction (p53, BRCA, WT1) as ovarian cancer and can also show an increased expression of HER-2/neu.

310. C

BRCA 1 -17q

BRCA 2- 13 q

311. C

Among candidates for metastasis oncogenes, best recognised are SNAIL andTWIST, which encode transcription factors whose primary function is to promote epithelial-to-mesenchymal transition (EMT). In EMT, carcinoma cells downregulate certain epithelial markers (e.g., E-cadherin) and upregulate certain mesenchymal markers (e.g., vimentin and smooth muscle actin). These changes are believed to favor the development of a promigratory phenotype that is essential for metastasis. Loss of E-cadherin expression seems to be a key event in EMT, and SNAIL and TWIST are transcriptional repressors that downregulate E-cadherin expression. EMT has been documented mainly in breast cancers; whether it is a general phe

312. C

In some tumors, connective tissue is scant and so the neoplasm is soft and fleshy. In other cases, the parenchymal cells stimulate the formation of an abundant collagenous stroma, referred to as desmoplasia. Some demoplastic tumors—for example, some cancers of the female breast—are stony hard or scirrhous

313. A, B,C

Microbe	Associated cancer
EBV	Burkitt lymphoma, Hodgkin lymphoma, nasopharyngeal carcinoma, 1° CNS lymphoma (in immunocompromised patients)
HBV, HCV	Hepatocellular carcinoma
HHV-8	Kaposi sarcoma
HPV	Cervical and penile/anal carcinoma (types 16, 18), head and neck cancer
H pylori	Gastric adenocarcinoma and MALT lymphoma
HTLV-1	Adult T-cell leukemia/lymphoma
Liver fluke (Clonorchis sinensis)	Cholangiocarcinoma
Schistosoma haematobium	Bladder cancer (squamous cell)

314. B

The true extent of chromosome rearrangements in cancer is only now coming into view thanks to sequencing of entire cancer cell genomes, which allows for comprehensive "reconstruction" of chromosomes from DNA sequences. Genomic sequencing has revealed not only many simple rearrangements (e.g., small deletions, duplications, or inversions) that were not appreciated by prior methods, but also much more dramatic chromosome "catastrophes" termed chromothrypsis (literally, chromosome shattering). Chromothrypsis is observed in 1% to 2% of cancers as a whole, but is found in up to 25% of osteosarcomas and other bone cancers and at relatively high frequency in gliomas as well. It appears to result from a single event in which dozens to hundreds of chromosome breaks occur within part or across the entirety of a single chromosome or several chromosomes

315. B

Hypercalcemia is the most common paraneoplastic syndrome.

But cancer associated hypercalcemia isn't always paraneoplastic. When the cancer produces some calcemic substances, only then you call it paraneoplastic.

Hypercalcemia due to primary or secondary involvement of skeleton isn't a paraneoplastic hypercalcemia.

Now cancer associated hypercalcemia can be due to different causes (paraneoplastic or otherwise). Three major mechanisms of hypercalcemia in tumors are as follows-

1. Osteolysis - either due to a primary tumor like myeloma or a secondary tumor like breast cancer.
2. Excessive 1,25(OH)2 D production like in lymphomas
3. True paraneoplastic conditions created by secretion of PTH related peptide (PTHrP) by tumors like squamous cell carcinoma lung

Clinical Syndromes	Major Forms of Underlying Cancer	Causal Mechanism
Endocrinopathies		
Cushing syndrome	Small-cell carcinoma of lung Pancreatic carcinoma Neural tumors	ACTH or ACTH-like substance
Syndrome of inappropriate antidiuretic hormone secretion	Small-cell carcinoma of lung Intracranial neoplasms	Antidiuretic hormone or atrial natriuretic hormones
Hypercalcemia	Squamous cell carcinoma of lung Breast carcinoma Renal carcinoma Adult T-cell leukemia/lymphoma	Parathyroid hormone-related protein (PTHRP), TGF-α, TNF, IL-1

Hypoglycemia	Ovarian carcinoma Fibrosarcoma Other mesenchymal sarcomas	Insulin or insulin-like substance
Polycythemia	Renal carcinoma Cerebellar hemangioma Hepatocellular carcinoma	Erythropoietin
Nerve and Muscle syndromes		
Myasthenia	Bronchogenic carcinoma Thymic neoplasms	Immunologic
Disorders of the central and peripheral nervous system	Breast carcinoma	
Dermatologic Disorders		
Acanthosis nigricans	Gastric carcinoma Lung carcinoma Uterine carcinoma	Immunologic; secretion of epidermal growth factor
Dermatomyositis	Bronchogenic carcinoma Breast carcinoma	Immunologic
Osseous, Articular, and Soft Tissue Changes		
Hypertrophic osteoarthropathy and clubbing of the fingers	Bronchogenic carcinoma Thymic neoplasms	Unknown
Vascular and Hematologic Changes		
Venous thrombosis (Trousseau phenomenon)	Pancreatic carcinoma Bronchogenic carcinoma Other cancers	Tumor products (mucins that activate clotting)
Disseminated intravascular coagulation	Acute promyelocyte leukemia Prostatic carcinoma	Tumor products that activate clotting
Nonbacterial thrombotic endocarditis	Advanced cancers	Hypercoagulability
Red cell aplasia	Thymic neoplasms	Unknown

316. B

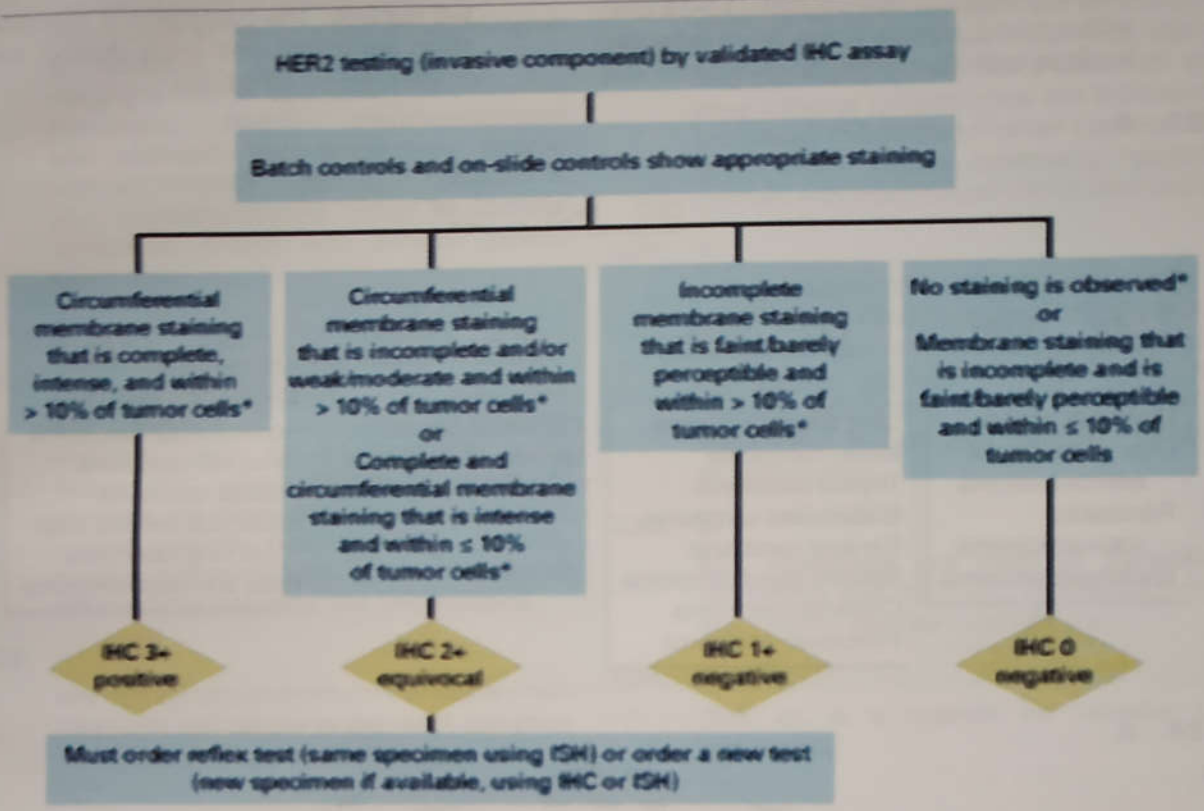
Cairo-Bishop Definition of Laboratory Tumor Lysis Syndrome

Element	Value	Change from Baseline
Uric acid	≥ 476 micro mol/L (8 mg/dL)	25% increase
Potassium	≥ 6.0 m mol/L (or 6 mEq/L)	25% increase
Phosphorous	≥2.1 mmol/L (6.5 mg/dL) for children or ≥ 1.45 mmol/L (4-5 mg/dL) for adults	25%increase
Calcium	≥1.75 mmol/L (7 mg/dL)	25%decrease

NOTE: Two of more laboratory changes within the period from 3 days before to 7 days after cytotoxic therapy are required to establish the diagnosis of tumor lysis syndrome.

317. B

Algorithm for evaluation of human epidermal growth factor receptor 2 (HER2) protein expression by immunohistochemistry (IHC) assay of the invasive component of a breast cancer specimen.


HER2 testing (invasive component) by validated IHC assay
Batch controls and on-slide controls show appropriate staining
Circumferential membrane staining that is complete, intense, and within > 10% of tumor cells*
Circumferential membrane staining that is incomplete and/or weak/moderate and within > 10% of tumor cells*
or
Complete and circumferential membrane staining that is intense and within ≤ 10% of tumor cells*
Incomplete membrane staining that is faint/barely perceptible and within > 10% of tumor cells*
No staining is observed*
or
Membrane staining that is incomplete and is faint/barely perceptible and within ≤ 10% of tumor cells
IHC 3+ positive
IHC 2+ equivocal
IHC 1+ negative
IHC 0 negative
Must order reflex test (same specimen using ISH) or order a new test (new specimen if available, using IHC or ISH)

318. A

Fixatives used in pathology

1. Histopathology routine- 10% neutral buffered formalin (most commonly used fixative)
2. Electron microscopy- glutaraldehyde
3. Testicular biopsies- Bouin's fluid (can be used for GIT and endocrine too)
4. Lymph node/bone marrow/kidney- Zenker's (good nuclear details) or B5
5. To dissolve fat for lymph node examination in radical resections- Carnoy's (also for karyotyping)
6. Cytopathology - Ethanol (95%)

319. A, B, C, D

Neoplasms which express neuroendocrine markers (synaptophysin, chromogranin A, NCAM / CD56 or protein gene product (PGP))

- Synaptophysin is strongly and diffusely expressed in most tumors
- Chromogranin A is focal / patchy
- CD56 and PGP are considered less specific
- Site specific markers are PDX1 and ISL1

320. A

Desmin- muscle origin

321. B

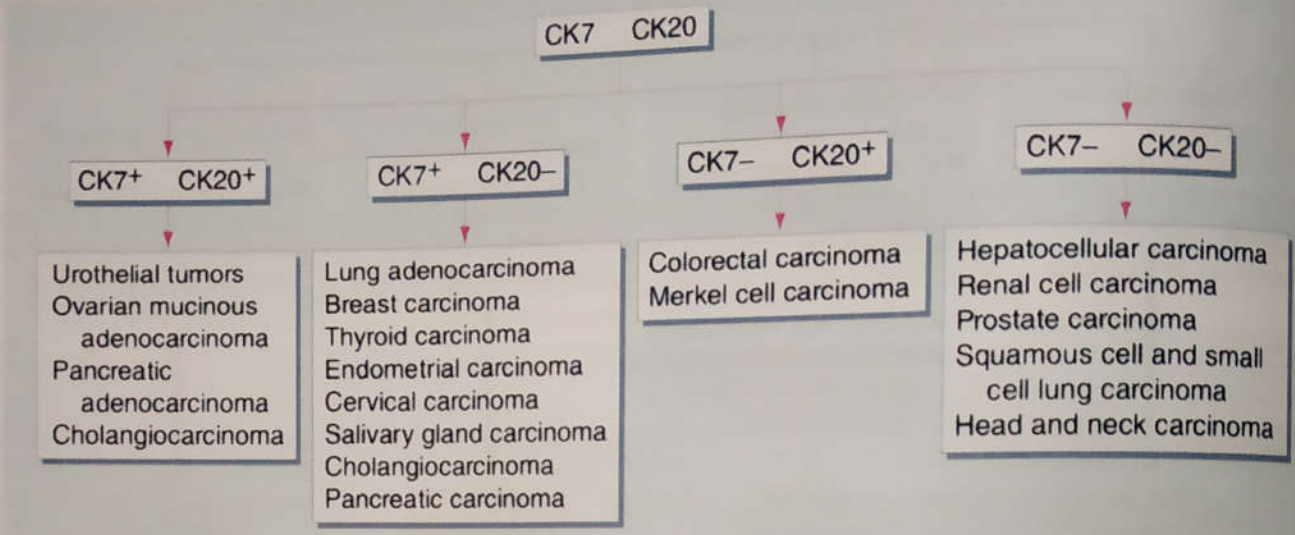
Three lymphotropic viruses—human T-cell leukemia virus-1 (HTLV-1), Epstein-Barr virus (EBV), and Kaposi sarcoma herpesvirus/human herpesvirus-8 (KSHV/HHV-8)—have been implicated as causative agents in particular lymphomas. HTLV-1 is associated with adult T-cell leukemia/ lymphoma. EBV is found in a subset of Burkitt lymphoma, 30% to 40% of Hodgkin lymphoma (HL), many B-cell lymphomas arising in the setting of T-cell immunodeficiency, and rare NK-cell lymphomas. In addition to Kaposi sarcoma. KSHV is associated with an unusual B-cell lymphoma that presents as a malignant effusion, often in the pleural cavity

322. 22 A

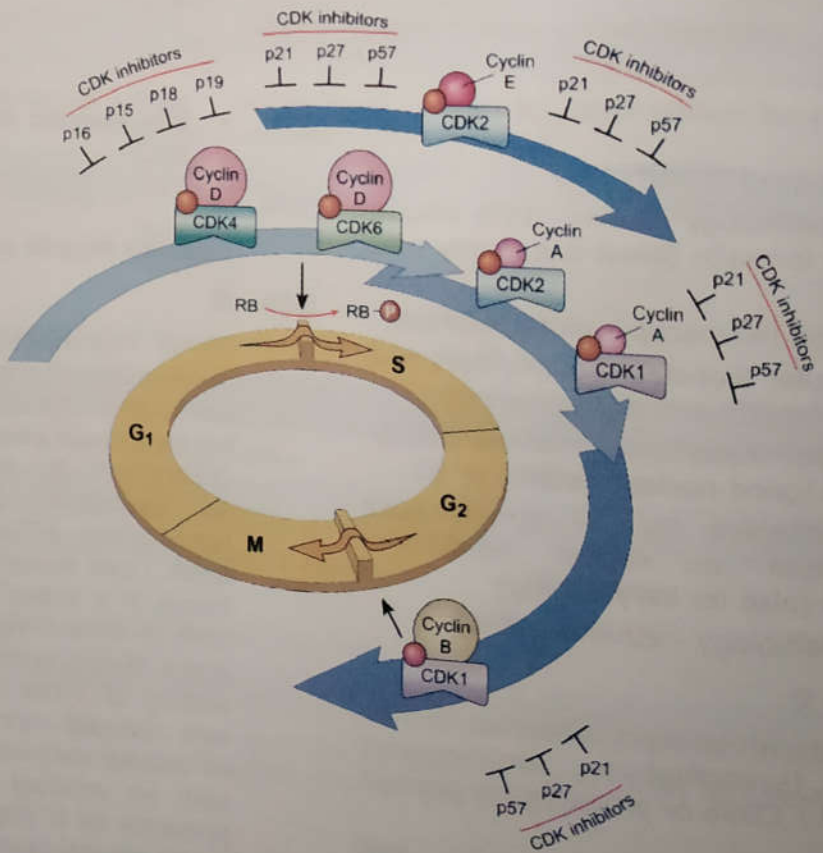
The megakaryoblasts are usually

medium-sized to large blasts (12-18 μm) with a round, slightly irregular, or indented nucleus with fine reticular chromatin and 1-3 nucleoli. The cytoplasm is basophilic, often agranular, and show distinct blebs or pseudopod formation.

323. A



324. A



325. B

Due to BCR ABL1 fusion in CML, the new protein formed is 210 kD or p210. P190 is seen in ALL.

326. B

Integrins are components of ECM which help in binding of cells to ECM. Although implicated in metastasis, E and N

cadherins (part of desmosomes) are more recognised in the mechanism of invasion of metastasis, the former, when lost, playing a role in epithelial-mesenchymal transition. Matrix metalloproteases and cathepsin help breakdown stroma and aid invasion. Loss of E cadherin is also characteristically noted in Lobular carcinoma breast and diffuse gastric carcinoma.

327. A

DCIS shows nuclear pleomorphism just like an invasive carcinoma. But the presence of basement membrane invasion, loss of myoepithelial cells favour IDC whereas central areas of necrosis are more characteristic of DCIS (called comedo type of DCIS). Hence, nuclear pleomorphism does not help to differentiate between the two entities.

328. A

Warthin Lynch syndrome or Hereditary non polyposis coli cancer is the most common familial cause of colorectal carcinoma. Patients affected are younger and usually involve the right colon. Mucinous, signet ring cell features are more common and intra tumoral lymphocytes are increased. Increased incidence of other cancers like endometrial (most common), gastric, ovarian and transitional cell is also noted.

329. A

Unfortunately, it is almost impossible to predict the clinical course of a carotid body tumor—mitoses, pleomorphism, and even vascular invasion are not reliable indicators. Hence, presence of metastasis is the only reliable indicator of malignancy.

330. C

RET mutations are seen in medullary carcinoma and RET/PTC rearrangements are seen in papillary carcinoma.

331. C

SOX 11 is a marker for mantle cell lymphoma.

332. D Ataxia telangiectasia

Disease	Inheritance	Chromosomal Location	Gene/Protein	Normal Function/Manifestation of Loss
Ataxia-telangiectasia	AR	11 q22.3	ATM/AIM	DNA repair after radiation injury/ neurologic and vascular lesions
Nevoid basal cell carcinoma syndrome	AD	9q22	PTCtf/PTCH	Developmental patterning gene/ multiple basal cell carcinomas; medulloblastoma, jaw cysts
Cowden syndrome	AD	10q23	PTENIPTEN	Lipid phosphatase/benign follicular appendage tumors (trichilemmomas); internal adenocarcinoma (often breast or endometrial)
Familial melanoma syndrome	AD	9p21	CDWV2/p16/ INK4 CDMV2/ p14/ARF	Inhibits CDK4/6 phosphorylation of RB, promoting cell cycle arrest/ melanoma; pancreatic carcinoma Binds MDM2, promoting p53 function/ melanoma; pancreatic carcinoma
Muir-Torre syndrome	AD	2p22 3p21	MSH2/ MSH2 MLH1IMLH1	Involved in DNA mismatch repair/ sebaceous neoplasia; internal malignancy (colon and others)
Neurofibromatosis I	AD	17q11	NF1/ neurofibromin	Negatively regulates RAS signaling/ neurofibromas
Neurofibromatosis II	AD	22q12	NF2/merlin	Integrates cytoskeletal signaling/ neurofibromas and acoustic neuromas

Tuberous sclerosis	AD	9q34 16p13	TSC1/hamartin TSC2/tuberin	Work together in a complex that negatively regulates mTOR/ angiofibromas/ mental retardation
Xeroderma pigmentosum	AR	9q22 and others	XPA/XPA and others	Nucleotide excision repair/melanoma and nonmelanoma skin cancers

333. B

Because of their chemical structures, some carcinogens interact preferentially with particular DNA sequences or bases, and thus produce mutations that are clustered at "hotspots" or that are enriched for particular base substitutions. One illustrative example of a chemical carcinogen associated with a mutational "hotspot" is aflatoxin B1, a naturally occurring agent

produced by some strains of a mold called Aspergillus. Aspergillus grows on improperly stored grains and nuts, and there is a strong correlation between the dietary level of this food contaminant and the incidence of hepatocellular carcinoma in parts of Africa and the Far East. Interestingly, aflatoxin B1

-associated hepatocellular carcinomas tend to have a particular mutation in TP53, a G : C→T : A transversion in codon 249 that produces an arginine to serine substitution in the p53 protein. In contrast, TP53 mutations are infrequent in liver tumors from areas where aflatoxin contamination of food does not occur, and few of these mutations involve codon 249. Similarly, lung cancers associated with smoking have a 10-fold higher mutational burden on average than lung cancers in nonsmokers, and these excess mutations are strongly skewed toward particular base substitutions known to be caused by carcinogens in cigarette smoke (the proverbial "smoking gun").

334. B

The UV portion of the solar spectrum can be divided into three wavelength ranges: UVA (320-400 nm), UVB (280-320 nm), and UVC (200-280 nm). Of these, UVB is believed to be responsible for the induction of cutaneous cancers. UVC, although a potent mutagen, is not considered significant because it is filtered out by the ozone layer surrounding the earth (hence the concern about ozone depletion).

335. C

Gene(s)	Function	Tumor (Approximate Frequency of Mutation)
DNMT3A	DNA methylation	Acute myeloid leukemia (20%)
MLL1	Histone methylation	Acute leukemia in infants (90%)
MLL2	Histone methylation	Follicular lymphoma (90%)
CREBBP/ EP300	Histone acetylation	Diffuse large B cell lymphoma (40%)
ARID1A	Nucleosome positioning/chromatin remodeling	Ovarian clear cell carcinoma (60%), endometrial carcinoma (30%-40%)
SNF5	Nucleosome positioning/chromatin remodeling	Malignant rhabdoid tumor (100%)
PBRM1	Nucleosome positioning/chromatin remodeling	Renal carcinoma (30%)

336. C

Malignancy	Translocation	Affected Genes
Chronic myelogenous leukemia (CML)	(9;22)(q34;q11)	ABL 9q34 BCR 22q11
Acute myeloid leukemia (AML)	(8;21)(q22;q22) (15;17)(q22;q21)	AML 8q22 ETO 21q22 PML 15q22 RARA 17q21
Burkitt lymphoma	(8;14)(q24;q32)	MKC 8q24 /Gtf 14q32
Mantle cell lymphoma	(11;14)(q13;q32)	CCND111 q13 IGH14q32
Follicular lymphoma	(14;18)(q32;q21)	IGH 14q32 BCL2 18q21
Ewing sarcoma	(11;22)(q24;q12)	FLI1 11 q24 EWSR1 22q12
Prostatic adenocarcinoma	(7:21)(p22;q22) (17:21)(p21;q22)	TMPRSS2 (21 q22.3) ETV1 (7p21.2) ETV4 (17q21)

337. A

An important emerging concept is that tumor cells actively inhibit tumor immunity by engaging normal pathways of immune regulation that serve as "checkpoints" in immune responses. Through a variety of mechanisms, tumor cells may downregulate the expression of costimulatory factors on antigen-presenting cells, such as dendritic cells; as a result, the antigen presenting cells fail to engage the stimulatory receptor CD28 and instead activate the inhibitory receptor CTLA-4 on effector T cells. This not only prevents sensitization but also may induce long-lived unresponsiveness in tumor-specific T cells. Tumor cells also may upregulate the expression of PD-L1 and PD-L2, cell surface proteins that activate the programmed death-1 (PD-1) receptor on effector T cells. PD-1, like CTLA-4, may inhibit T cell activation. Antibodies that block the inhibitory CTLA-4 or PD-1 receptors have produced promising results in clinical trials conducted in patients with advanced-stage solid tumors. Additional clinical trials are being planned using both PD-1 and CTLA-4 blocking antibodies in combination with each other and with conventional or targeted chemotherapy. The success of these agents has led to a new paradigm in cancer immunotherapy, sometimes called "checkpoint blockade". This is centered on the idea that treatments that remove the "brakes" imposed by tumors on host anti-tumor immune responses can be highly effective in treating cancer.

338. D

339. A

340. C

341. D

342. D

343. A

344. A

345. A

346. A, B, C

347. D

348. A

349. D

350. D
351. A
352. A
353. B
354. B
355. C
356. B
357. A
358. C
359. B
360. D
361. D
362. B
363. c

Red Blood Cells and its Disorders – Questions

364. What should be the sequence of events during collection of blood sample?

a. Ask the patient his name verify from file → collect blood → Label the sample at bedside
b. Look at the file → Collect sample → Label the sample at bedside
c. Pre label the sample vials → Check the file patient details → Collect sample
d. Collect sample → Confirm name from file → Label the sample vial

365. Which vacutainer is used for electrolyte estimation?

a. Na Citrate
b. EDTA
c. Fluoride
d. Lithium heparin

366. What does the red cell distribution width (RDW) deal with?

a. Hypochromia
b. Anisocystosis
c. Poikilocytosis
d. Anisochromia

367. A 25-years-old female came to OPD 1 year after postpartum. She was treated for iron deficiency anemia while pregnancy. Now she is pale and her HB was 5% and reticulocyte count was 9%. Her corrected retic count is?

a. 6
b. 4.5
c. 3
d. 1

368. A 26-years-old patient having RBC count 2 lakhs/mm³, PCV 30% and haemoglobin 9.3, What is the type of anemia?

a. Iron deficiency anemia
b. Folic acid deficiency anemia
c. Thalassemia
d. Sideroblatic anemia

369. Spur cells are seen in:

a. Hereditary spherocytosis
b. G6PD deficiency
c. Liver disease
d. Sideroblastic anemia

370. Supravital staining is used for?

a. Nucleated RBCs
b. Reticulocytes
c. Basophils
d. Myeloblasts

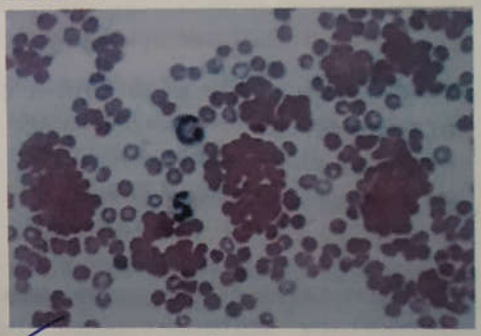
371. Howel-Jolly bodies may be seen after-

a. Hepatectomy
b. Splenectomy
c. Pancreatectomy
d. Cholecystectomy

372. All are true about polycythemia vera except?

a. Increased ESR
b. Decreased erythropoietin
c. Increased LAP score
d. Increased blood volume

373. A 25-years-old female presented in December month with chronic fatigue and cyanosis with bluish lips and arthralgia. Peripheral blood film is shown below. What is the likely cause?



a. Cold AIHA
b. Warm AIHA
c. Hemoglobinopathy
d. G6PD Deficiency

364. a	665. d	366. b	367. c	368. a	369. c	370. b	371. b	372. a	373 a

374. Which of the following is absent in haemolytic anemia? (PGI)

a. Increased indirect bilirubin
b. Increased direct bilirubin
c. Increased reticulocyte count
d. Jaundice
e. Increased LDH

375. Which one of the following is not associated with a high reticulocyte count?

a. Acute bleed
b. Haemolytic anemia
c. Megaloblastic anemia
d. Response to treatment in 'nutrition – deficiency anemia

376. Adult hemoglobin consists of the following chains?

a. $2\alpha + 2\beta$
b. $2\alpha + 2\delta$
c. $2\beta + 2\Upsilon$
d. $2\alpha + 2\Upsilon$

377. Direct coomb's test is positive in haemolytic anemia due to:

a. Paroxysmal cold hemoglobinuria
b. Paroxysmal nocturnal hemoglobinuria
c. Idiopathic thrombocytopenic purpura
d. Hemolytic uremic syndrome

378. Bart hemoglobin is tetramer of:

a. α chain
b. β chain
c. Υ chain
d. δ chain

379. A 29-years-old female who has hereditary spherocytosis and mild anemia has developed a low grade fever with malaise. After a week she feels very tired and appears pale. Her haematocrit drops from the usual value of 36% to 28%. There is no change in the appearance of red cells morphology. Reticulocytes are absent from the following events has most likely occurred:

a. Development of anti-red cell antibodies
b. Disseminated intravascular coagulopathy
c. Accelerated extravascular hemolysis in the spleen
d. Reduced erythropoiesis from parvo virus infection

380. CD59 deficiency leads to:

a. PNH
b. Chediak-Higashi disease
c. Hairy cell leukemia
d. Hemolytic uremic syndrome

381. All decrease in iron deficiency anemia except?

a. Ferritin
b. TIBC
c. Iron
d. Transferrin saturation

382. Which of these is not involved in iron metabolism?

a. Hepcidin
b. Ferroportin
c. Transthyretin
d. Ceruloplasmin

383. Which of the following is accurate regarding the internal iron homeostasis in iron deficiency anemia:

a. Transferrin receptor 1-iron responding elements increases transferrin receptor mRNA concentration and synthesis
b. Ferritin mRNA concentration – iron response element increases and ferritin synthesis decreases
c. Ferritin mRNA concentration – iron response element decreases and ferritin synthesis increases
d. Transferrin receptor 1-iron responding elements decreases transferrin receptor mRNA concentration and increases synthesis

374. b	375. c	376. a	377. a	378. c	379. d	380. a	381. b	382. c	383.

384. Match List-I with List-II and select the correct answer using the code given below the Lists?

Code:

	A	B	C	D
a.	1	4	2	3
b.	2	3	4	1
c.	4	1	2	3
d.	3	1	2	4

385. Criteria for severe aplastic anemia are all except?

a. BM cellularity >25%
b. Reticulocyte < 60,000/ul
c. Platelet < 20,000/ul
d. Absolute neutrophil count < 1500/ul

386. 2 years-old child presents with short stature and café-au-lait spots. Bone marrow aspiration yields a little material and mostly containing fat. What is your diagnosis:

a. Fanconi anemia
b. Dyskeratosis congenita
c. Tuberous sclerosis
d. Osteogenesis imperfecta

387. Dry tap is a feature:

a. Anemia of chronic disease
b. Megaloblastic anemia
c. Aplastic anemia
d. Sickle cell anemia

388. Erythropoeitin acts on

a. CFU
b. BFU
c. Pro erythroblast
d. Reticulocyte

389. Intravascular haemolytic anemia is seen in all except-

a. Autoimmune haemolytic anemia
b. TTP
c. Thalassemia
d. Infection

390. Shape of RBC is biconcave due to-

a. Ankyrin
b. Spectrin
c. Band protein
d. Glycophorin-C

391. Not true about hereditary spherocytosis-

a. Defect in ankyrin
b. Decreased MCV
c. Decreased MCHC
d. Reticulocytosis

392. A 23-years-old female presented with jaundice and pallor for 2 months. Her peripheral blood smear shows the presence of spherocytes. The most relevant investigation to arrive at a diagnosis is-

a. Reticulocyte count
b. Osmotic fragility test
c. Coombs' test
d. Tests for PNH

393. What is affected in HBS (Hemoglobin S)

a. Stability
b. Function
c. Affinity
d. Solubility

394. A 6 years old Afro-American boy develops recurrent infections, severe abdominal pain, and a non-healing ulcer over left foot. RBC has abnormal shape on microscopy. Which of the following is correct?

a. Trinucleotide repeat mutation
b. Single base substitution
c. Antibodies against RBC membrane
d. Genomic imprinting

395. Autosplenectomy is seen in-

a. Hereditary spherocytosis
b. G6PD deficiency
c. Sickle cell anemia
d. Thalassemia major

396. One of the common variants of sickle cell disease is frequently marked by lesser degree of haemolytic anaemia and greater propensity for the development of retinopathy and aseptic necrosis of bones:

a. Sickle cell trait
b. HBSC disease
c. Sickle thalassemia
d. Sickle HbE disease

384. c	385. d	386. a	387. c	388. a	389. c	390. b	391. c	392. c	393 d
394. b	395. c	396. b							

397. In α-thalassemia- hemolysis is due to

a. Excess α-chain
b. No α-chain
c. Excess β-chain
d. No β-chain

398. Thalassemia gives protection against-

a. Filaria
b. Kala azar
c. Malaria
d. Leptospirosis

399. Paroxysmal nocturnal hemoglobinuria is due to-

a. Acquired red cell defect
b. Congenital red cell defect
c. Autoimmune defect
d. Lead poisoning

400. Which is not a feature of paroxysmal nocturnal hemoglobinuria?

a. Increased LAP score
b. Thrombosis
c. Thrombocytopenia
d. Hemolysis

401. Cold agglutinin is-

a. IgG
b. IgM
c. IgA
d. IgD

402. HbH disease is associated with-

a. Deletion of 3 alpha genes
b. Deletion of 4 alpha genes
c. Deletion of 3 beta genes
d. Deletion of 4 beta genes

403. Red cells containing granules of non-haem iron which gives positive Prussian blue reaction with Perl's stain as well as stain with Romanowsky dyes (referred to as pappenheimer bodies) are known as-

a. Schistocytes
b. Spherocytes
c. Sideroblasts
d. Siderocytes

404. Earliest manifestation of megaloblastic anemia-

a. Macrocytosis
b. Hypersegmented neutrophils
c. Basophilic stippling
d. Cabot ring formation

405. The best suited anticoagulant for osmotic fragility test is-

a. Heparin
b. EDTA
c. Trisodium citrate
d. Potassium oxalate

406. Causes of a dry tap when performing a bone marrow aspiration may be expected in:

a. Iron deficiency anemia
b. Refractory anemia with ringed sideroblasts
c. Hairy cell leukemia
d. Hereditary spherocytosis

407. In a patient with β thalassemia trait, all of the following features are typically observed except:

a. Significantly high RDW
b. Mild microcytic hypochromic anemia
c. Target cells on the peripheral smear
d. Disproportionately elevated RBC count

408. A 30-years-old male with long-standing history of diarrhea, has macrocytosis, Howell-Jolly bodies in RBC, target cells and small shrunken spleen. The most likely diagnosis is:

a. Sickle cell disease
b. Celiac disease
c. Crohn's disease
d. Liver disease

409. Iron deficiency may result in the misdiagnosis of:

a. Sickle cell trait
b. Sickle cell disease
c. β thalassemia trait
d. β thalassemia disease

397. c	398. c	399. a	400. a	401. b	402. a	403. d	404. a	405. a	406. c
407. a	408. d	409. c							

410. A child with a known diagnosis of hereditary spherocytosis develops "slapped cheek" appearance as well as profound anemia. Reticulocyte response to the anemia is inadequate. A bone marrow examination is most likely to demonstrate:

a. Hypocellular marrow with reduction of all cell lines
b. Erythroid hyperplasia as a response to acute hemolysis
c. Reduced erythropoiesis with giant erythroblasts and intranuclear inclusions
d. "Owl eye" appearance, due to CMV infection in erythroid precursors

411. All statements regarding hereditary spherocytosis is true except:

a. May be diagnosed by flow cytometry
b. May be diagnosed by incubated osmotic fragility test
c. When inherited, most cases are autosomal recessive
d. The direct antiglobulin test (DAT) is typically negative

412. All of the following statements regarding paroxysmal nocturnal hemoglobinuria (PNH) are true except:

a. PNH is transmitted as X-linked recessive
b. PNH may be diagnosed by flow cytometry
c. PNH may be diagnosed by Ham's acidified serum test
d. Occurs due to mutation of the PIG-A gene

413. Spiculated RBCs with evenly spaced spikes are called:

a. Acanthocytes
b. Stomatocytes
c. Echinocytes
d. Dacryocytes

414. Which of the following indicates intravascular hemolysis?

a. Target cells
b. Schistocytes
c. Acanthocytes
d. Basophilic stippling

415. Pappenheimer bodies are composed of?

a. Copper
b. Zinc
c. Iron
d. Lead

416. Blister cells seen in which condition?

a. G6PD deficiency
b. Thalassemia
c. Sickle cell anemia
d. AIHA

417. Most common mutation in hereditary elliptocytosis:

a. Spectrin
b. Ankyrin
c. Glycophorin A
d. Band 4.2

418. In hereditary spherocytosis, which gene is altered?

a. Spectrin
b. Laminin
c. Desmin
d. Vimentin

419. Which of the following tests is gold standard to diagnose a case of PNH?

a. HAMS test
b. Sucrose lysine test
c. Flow cytometry
d. Bone marrow

420. Which of the following are true regarding a child with iron deficiency anaemia:

a. Raised MCV
b. Raised transferrin saturation
c. Increased TIBC
d. Increased ferritin

421. Following are causes of megaloblastic anemia except?

a. Defect in DNA synthesis
b. Folic acid deficiency
c. Lead toxicity
d. Vit. B12 deficiency

410. c	**411. c**	**412. a**	**413. c**	**414. b**	**415. c**	**416. a**	**417. a**	**418. a**	**419. c**
420. c	**421. c**								

422. Iron metabolism and regulation are important for RBC precursor cell. Which of the following helps in regulation of iron metabolism but is not specific for iron?

a. Hepcidin
b. DMT-1
c. Ferroportin
d. Ferritin

423. Transfer of Iron from enterocyte to plasma is inhibited by?

a. Hepcidin
b. DMT-1
c. Ferroportin
d. Haphaestin

424. Parvovirus preferentially involves in which of the following cells:

a. Erythroid progenitors
b. Myeloid precursors
c. Megakaryocytes
d. Both a & c

425. Most common tumor associated with pure red cell aplasia:

a. Hepatoma
b. Hodgkins lymphoma
c. Thymoma
d. Bronchogenic carcinoma

426. Anemia with reticulocytosis is seen in-

a. Hemolysis
b. Iron deficiency anemia
c. Vitamin B2 deficiency
d. Aplastic anemia

427. Progenitor hematopoietic stem cells originate in-

a. Bone marrow
b. Thymus
c. Lymph node
d. Spleen

428. Osmotic fragility is increased in

a. Sickle cell anemia
b. Thalassemia
c. Hereditary spherocytosis
d. Chronic lead poisoning

429. Most common cause of spherocytes -

a. Autoimmune haemolytic anemia
b. Vit B12 deficiency
c. Aplastic anemia
d. Heraditary spherocytosis

430. In sickle cell anemia all are seen except-

a. Sickle cells
b. Target cells
c. Howell Jolly bodies
d. Ringed sideroblast

431. Person having heterozygous sickle cell trait is protected from infection of-

a. Plasmodium falciparum
b. P. vivax
c. Pneumococcus
d. Salmonella

432. HbA2 is raised in-

a. Beta trait thalassemia
b. Sickle cell anemia
c. Hereditary spherocytosis
d. None

433.. In microangiopathic haemolytic anemia, cells seen are-

a. Schistocytes
b. Stomatocytes
c. Spherocytes
d. Anisocytes

434. Response to iron in iron deficiency anemia is denoted by-

a. Restoration of enzymes
b. Reticulocytosis
c. Increase in iron binding capacity
d. Increased in haemoglobin

435. Most common peripheral smear finding in "anemia of chronic disease" is-

a. Microcytic hypochromic
b. Normocytic normochromic
c. Macrocytic
d. None

436. A 40 years old male had undergone splenectomy 20 years ago. Peripheral blood smear Examination would show the presence of-

a. Dohle bodies
b. Hypersegmented neutrophils
c. Spherocytes
d. Howell-Jolly bodies

422. a	423. a	424. a	425. c	426. a	427. a	428. c	429. a	430. d	431. a
432. a	433. a	434. b	435. b	436. d					

437. The stain used for the demonstration of reticulocyte is-
a. Wright stain
b. Brilliant cresyl blue
c. Alcian blue
d. Giemsa stain

438. Microspherocytosis in peripheral blood smear are seen in-
a. Congenital spherocytosis
b. Autoimmune acquired hemolytic anemia
c. Thalassemia
d. All of the above

439. Which of the following is an effective marker for erythroid precursors?
a. CD34
b. CD71
c. CD41
d. CD61

440. The following are established causes of bone marrow granulomas:
a. Hodgkin lymphoma
b. Metastatic disease
c. Histoplasmosis
d. All of the above

441. In adults, bone marrow metastasis from the following primary tumors are common except:
a. Prostatic carcinoma
b. Meningioma
c. Bronchogenic carcinoma
d. Breast cancer

442. Peripheral blood findings most characteristic of metastatic tumor to the bone marrow is:
a. Leukoerythroblastic blood picture and presence of tear drop cells
b. Microcytic hypochromic red cells
c. Macrocytic anemia
d. Leukocytosis

443. The following are true about reticulocytes except:
a. They are young red cells and contain ribosomal RNA
b. Reticulocyte count is traditionally expressed as a percentage
c. Reticulocyte count is low in bone marrow failure patients
d. Reticulocyte count is low in haemolytic anemia patients

444. The following are all included in the differential diagnosis of microcytic hypochromic anemia except:
a. HbE disease
b. β thalassemia trait
c. Iron deficiency
d. HbS disease

445. In a patient with iron deficiency, which of the following features are an expected finding:
a. Minimal anisocytosis
b. Abundant target cells
c. Reduced transferrin iron binding capacity
d. Elevated levels of free erythrocyte protoporphyrin (FEP)

446. Anemia of chronic disease is characterized by all of these except:
a. Low levels of serum iron
b. Reduced transferrin saturation
c. Reduced iron binding capacity
d. Reduced serum ferritin

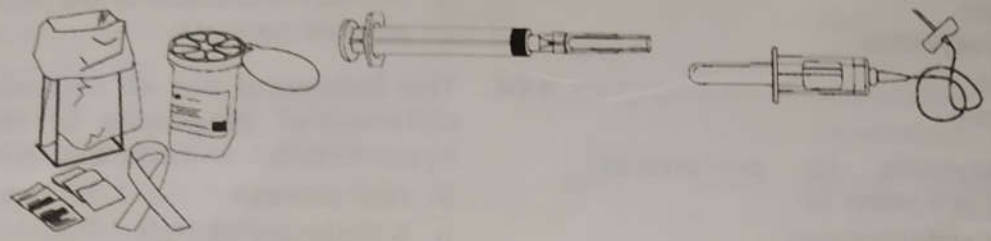
447. Bone marrow findings in megaloblastic anemia include all of the following except:
a. Nuclear cytoplasmic asynchrony of developing erythroid cells
b. Giant metamyelocytes
c. Hypersegmented megakaryocytes
d. Absent stainable iron in the bone marrow

448. A patient with known sickle cell disease registers with a new physician. CBC ordered by the patient's new physician demonstrates macrocytosis of the red blood cells. The most likely explanation is:
a. Iron deficiency
b. B12 deficiency
c. Folate deficiency
d. Patient is on hydroxyurea

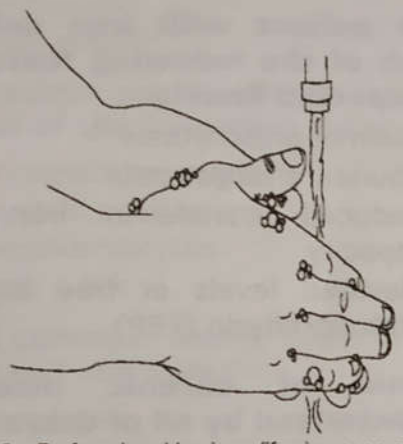
437. b	438. b	439. b	440. d	441. b	442. a	443. d	444. d	445. a	446. d
447. d	448. b								

364. A

WHO Guidelines

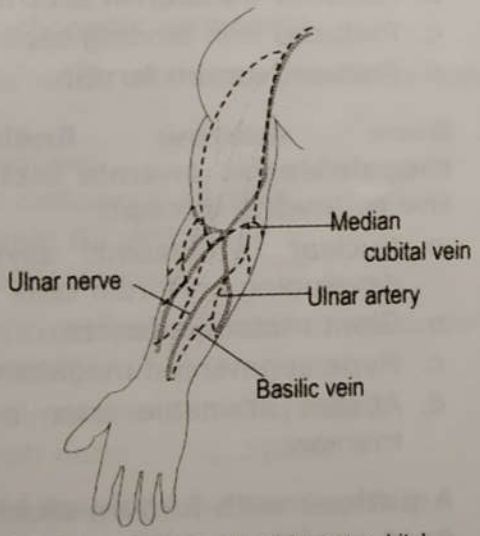


1. Assemble equipment and include needle and syringe or vacuum tube, depending on which is to be used.

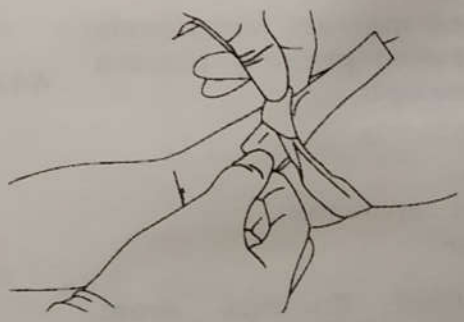


2. Perform hand hygiene (if using soap and water, dry hands with single-use towels).

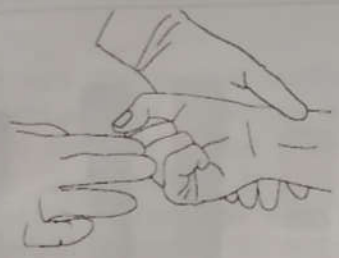
3. Identify and prepare the patient.



4. Select the site, preferably at the antecubital area (i.e. the bend of the elbow). Warming the arm with a hot pack, or hanging the hand down may make it easier to see the veins. Palpate the area to locate the anatomic landmarks. DO NOT touch the site once alcohol or other antiseptic has been applied.



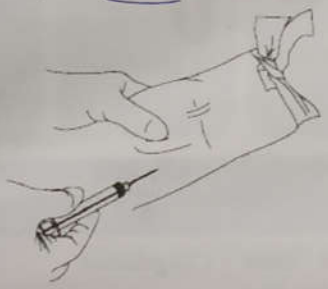
5. Apply a tourniquet, about 4–5 finger widths above the selected venepuncture site.



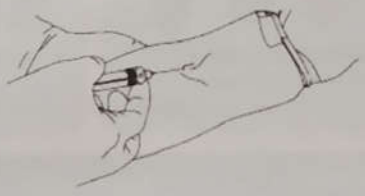
6. Ask the patient to form a fist so that the veins are more prominent.

7. Put on well-fitting, non-sterile gloves.

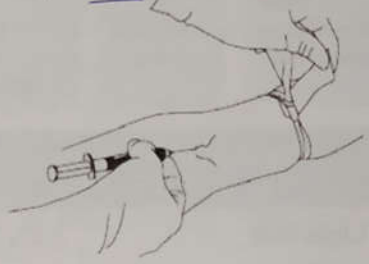
8. Disinfect the site using 70% isopropyl alcohol for 30 seconds and allow to dry completely (30 seconds).



9. Anchor the vein by holding the patient's arm and placing a thumb BELOW the venepuncture site.

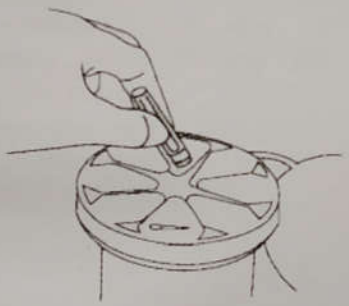


10. Enter the vein swiftly at a 30 degree angle.

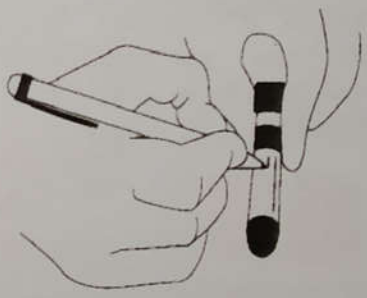


11. Once sufficient blood has been collected, release the tourniquet BEFORE withdrawing the needle.

12. Withdraw the needle gently and then give the patient a clean gauze or dry cotton-wool ball to apply to the site with gentle pressure.

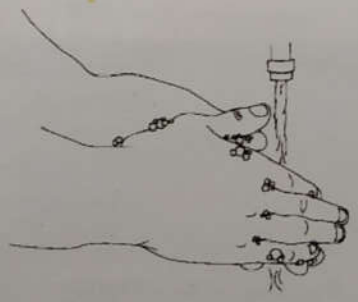


13. Discard the used needle and syringe or blood-sampling device into a puncture-resistant container.



14. Check the label and forms for accuracy.

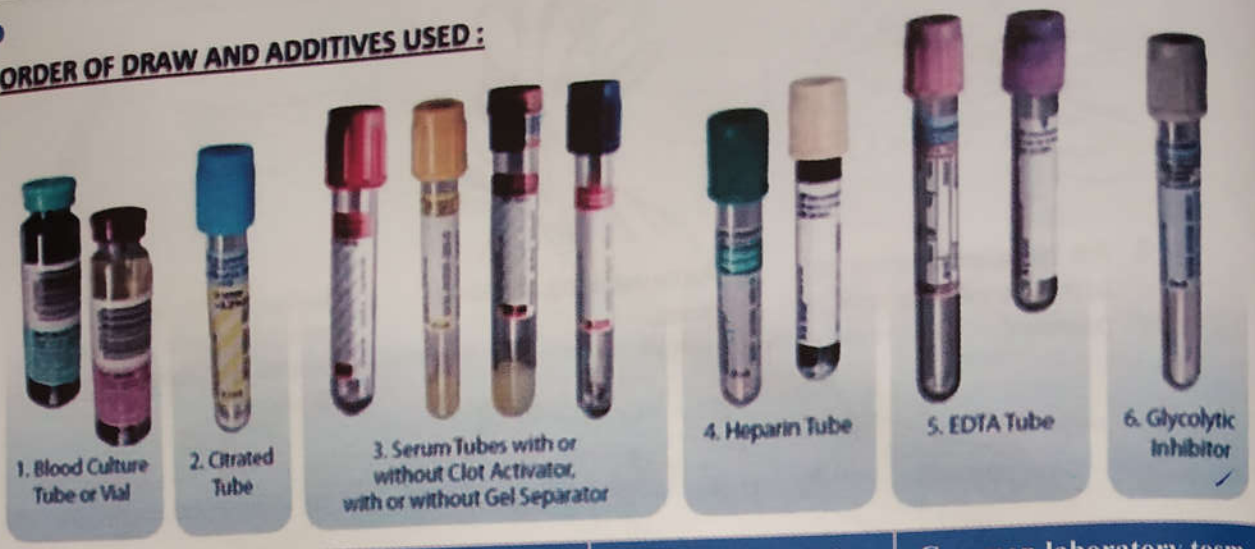
15. Discard sharps and broken glass into the sharps container. Place items that can drip blood or body fluids into the infectious waste.



16. Remove gloves and place them in the general waste. Perform hand hygiene. If using soap and water, dry hands with single-use towels.

365. D

ORDER OF DRAW AND ADDITIVES USED :


1. Blood Culture Tube or Vial
2. Citrated Tube
3. Serum Tubes with or without Clot Activator, with or without Gel Separator
4. Heparin Tube
5. EDTA Tube
6. Glycolytic Inhibitor

Tube cap color	Addltivo	Function of Addltive	Common laboratory tosm
Light blue	3.2% Sodlum citrate	Prevents blood from dotting by binding caldum	Coagulation
Red or gold (mottled or "tiger" top used with some tubes is not shown)	Serum tube with or without clot acwator or gel	Clot activator promotes blood clotting with glass or silica particles. Gel separates serum from cells.	Chemistry, serology, immunology
Green	Sodium or lithium heparin with or without gel	Prevents clotting by inhibiting thrombin and thromboplastin	Stat and routine chemistry
Lavender or pink	Potassium EDTA	Prevents clotting by binding calcium	Hematology and blood bank
Gray	Sodium fluoride, and sodium or potassium oxalate	Fluoride inhibits glycolysis. and oxalate prevents clotting by precipitating calcium.	Glucose (especially when testing will be delayed), blood aloohol, lactic acid

366.B

Red cell distribution width (RDW) is a parameter that measures variation in red blood cell size or red blood cell volume. RDW is elevated in accordance with variation in red cell size (anisocytosis), ie, when elevated RDW is reported on complete blood count, marked anisocytosis (increased variation in red cell size) is expected on peripheral blood smear review.

The reference range for RDW is as follows:

- RDW-SD 39-46 fL
- RDW-CV 11.6-14.6% in adult (usually followed)

Option A- means low MCHC (central pallor of the cell will be more than 2/3rd)

Option C- means variation in shape of RBCs; needs a peripheral smear examination to determine

Option D- means variation in chromicity; not a routinely used parameter

367.C

Corrected reticulocyte count = (reticulocyte % × hemoglobin)/15

368.A

Remember these formulae to calculate in questions what's missing

1. MCV = PCV/RBC x 10
2. MCH = Hb/RBC x10
3. MCHC = Hb/PCV x100

So here MCV= (30/2) x 10 = 150 which is macrocytic anemia and only one of the options is macrocytic, rest are microcytic anemias.

369.C

Terminology (Greek Meaning)	Old Terms, Synonyms	Description	Micrograph	Associated Diease States
Discocyte (disc)	Biconcave disc	Biconcave disc form of RBC		
Echinocyte (I-III) (sea urchin)	"Burr cell," crenated cell, "berry cell"	Spiculated RBC with short, equally spaced projections over entire surface; progressing from the "crenated disc" (echinocyte I) to the crenated sphere (echinocyte IV-not shown) with nearly complete loss of spicules		Uremia, liver disease Low-potassium red cells Immediately posttransfusion with aged or metabolically depleted blood Carcinoma of stomach and bleeding peptic ulcers
Acanthocyte (spike)	"Spur cell," acanthoid cell, acanthrocyte	Irregularly spiculated RBC with projections of varying length and position		Abetalipoproteinemia Alcoholic liver disease Postsplenectomy state Malabsorptive states
Stomatocyte (I-III) (mouth)	Mouth cell, cup form, mushroom cap, uniconcave disc, microspherocyte	Bowled-shaped RBC with single concavity; progressing from shallow bowl (I) to near sphere with small dimple (seen as mouth-shaped form in peripheral flm)		Hereditary spherocytosis Hereditary stomatocytosis Alcoholism, cirrhosis, obstruc- tive liver disease Erythrocyte sodium-pump defect

366.B

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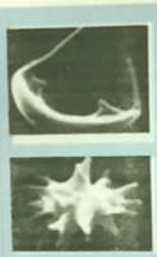
Spherostomatocyte (sphere)	Spherocyte, prelytic sphere, microspherocyte	Spherical RBC with dense hemo- globin content; scanning electron microscopy shows a persistent min- imal dimple		Hereditary (cells actu spheroston Immune h anemia Po Heinz bod anemia W hemolysis hemolysis
Schizocyte (cut)	Schistocyte, helmet cell, fragmented cell	Split RBC, often showing half-disc shape with two or three pointed extremities; may be small, irregular fragment		Microang hemolytic (TTP, DIC glomerulo renal graf Carcinom valve hem thetic or p valves) Se March her
Elliptocyte (oval)	Ovalocyte	Oval to elongated ellipsoid RBC (with polarization of hemoglobin)		Hereditary Thalassem defciency anemias M anemias
Drepanocyte (sickle)	Sickle cell	RBC containing polymerized hemo- globin S; showing varying shapes from bipolar, spiculated forms to holly-leaf and irregularly spiculated forms		Sickle cell (SS, S trai S thalasse Hemoglob Hemoglob
Codocyte (bell)	Target cell	Bell-shaped RBC that assumes a tar-get shape on dried flms of blood		Obstructi Hemoglo (S, C) Th Iron defci Postsplen
Dacryocyte (tear)	Teardrop cell	RBC with a single elongated or pointed extremity		Primary Myeloph Thalasse
Leptocyte (thin)	Thin cell, wafer cell	Thin, fat RBC with hemoglobin at periphery		Thalasse liver dise defciency
Keratocyte (horn)	Horn cell	RBC with spicules resulting from ruptured vacuole; cell appears half- moon shaped or spindle shaped		DIC or v prosthesi

spherocytosis ally natocytes) emolytic sttransfusion y hemolytic ater-dilution Fragmentation
opathic anemia , vasculitis, nephritis, t rejection) atosis Heart- olysis (pros- athologic vere burns noglobinuria
elliptocytosis ia Iron Myelophthisic Megaloblastic
disorders t, SC, SD, mia, etc.) bin C-Harlem bin Memphis/S
ve liver disease pinopathies alassemia ency ectomy state
myelofbrosis thisic anemias mia
mia Obstructive ase (± iron)
ascular s

370. B

Supravital stains like brilliant cresyl blue, new methylene blue and crystal violet are used in hematology to stain inclusions like Heinz bodies and HbH inclusions and the immature RBCs reticulocytes.

371. B

Common RBC Inclusions	Cartoon Image	Inclusion	May be associated with
Howell jolly Bodies		DNA	Hyposplenism Asplenism Severe hemolytic anemia
Heinz Bodies		Hemoglobin	G6PD deficiency Oxidant drugs Unstable hemoglobin
Pappenheimer Bodies		Iron deposits	Thalassemia SlderoblHtlc anemia Hemolytic anemia Post-splenectomy
Hemoglobin H Inclusion		Hemoglobin	Hemoglobin H disease
Basophilic Stippling		Ribosomes	Lead poisoning Thalusemia Sickle cell anemia MDS

372. A (single best answer)

The ESR is decreased in polycythemia, hyperviscosity, sickle cell anemia, leukemia, low plasma protein (due to liver or kidney disease) and congestive heart failure. Although increases in immunoglobulins usually increase the ESR, very high levels can reduce it again due to hyperviscosity of the plasma.

373. A

Cold agglutinin disease- mediated by IgM (thus causing agglutination as seen in the peripheral smear)

374. B

Hemolysis causes release of intra cellular enzymes like LDH thus leading to increased levels. Breakdown of Hb causes increased indirect bilirubin and jaundice. Compensatory hyperplasia in marrow causes increased reticulocyte counts for most hemolytic anemias.

375. C > A

376. A

Option B: HbA2

Option D: HbF

Hemoglobin	Chain Composition	Primary Site	Appearance
Gower-1	$\zeta_2\varepsilon_2$	Yolk sac	<5-6 weeks
Gower-2	$\alpha_2\varepsilon_2$	Yolk sac	4-13 weeks
Portland	$\zeta_2\gamma_2$	Yolk sac	4-13 weeks
Fetal (F)	$\alpha_2\gamma_2$	Liver	Early, 53-95% at term
Adult (A]	$\alpha_2\beta_2$	Marrow	9 weeks, 5-45% at term

377. A

PCH is an IgG mediated hemolytic anemia, which can be detected via Coombs test.

Option B- not an antibody mediated hemolytic anemia

Option C- not a hemolytic anemia- antibody mediated platelet destruction

Option D- microangiopathic hemolytic anemia

378. C

Seen in thalassemias

379. D

Parvovirus infection selectively attacks erythroid precursors thus reducing erythropoiesis and causing a dip in reticulocyte count and haematocrit.

380. A

Also known as Membrane inhibitor of reactive lysis (MIRL)

381. B

	Iron deficiency	Chronic disease	Hemochromatosis
Serum iron	↓	↓	↑
Transferrin or TIBC	↑	↓	↓
Ferritin	↓	↑	↑
% Transferrin saturation (serum iron/TIBC)	↓↓	–	↑↑

382. C

Option A- regulates iron levels by causing internalisation and proteolysis of ferroportin

Option B- channel for iron to leave the enterocyte in ferrous state, on the basolateral surface

Option D- helps in conversion of ferrous ion to ferric to bind to tran

383.

Transferrin receptor 1 - iron responding elements increases transferrin receptor mRNA concentration and synthesis.

	Decreasing iron	Increasing iron
Iron uptake	↑	↓
Transferrin receptor synthesis	↑	↓
Transferrin receptor mRNA concentration	↑	↓
Iron storsge	↓	↑
Ferritin synthesis	↓	↑
Ferritin mRNA concentration	No change	No change

384. C

List-I (Blood picture)	List-II (Type of Anemia)
Microcytic, hypochromic red cells	Vitamin B12 deficiency anemia
Macrocytic, hypochromic red cells	Thalassemia major
Large number of early, intermediate and late erythroblasts	Aplastic anemia
Low reticulocyte count	Iron-deficiency anemia

385. D

Classification	Criteria
Severe	BM cellularity < 25% (or < 50% if < 30% of BM is hematopoietic cells) AND ≥ 2 of the following: • Peripheral blood neutrophil count < 0.5 × 10^9/L • Peripheral blood platelet count < 20 × 10^9/L • Peripheral blood reticulocyte count < 20 × 10^9/L
Very severe	As above, but peripheral blood neutrophil count must be <0.2 × 10^9/L
Nonsevere	Hypocellullar BM with peripheral blood values not meeting criteriafor severe aplastic anemia

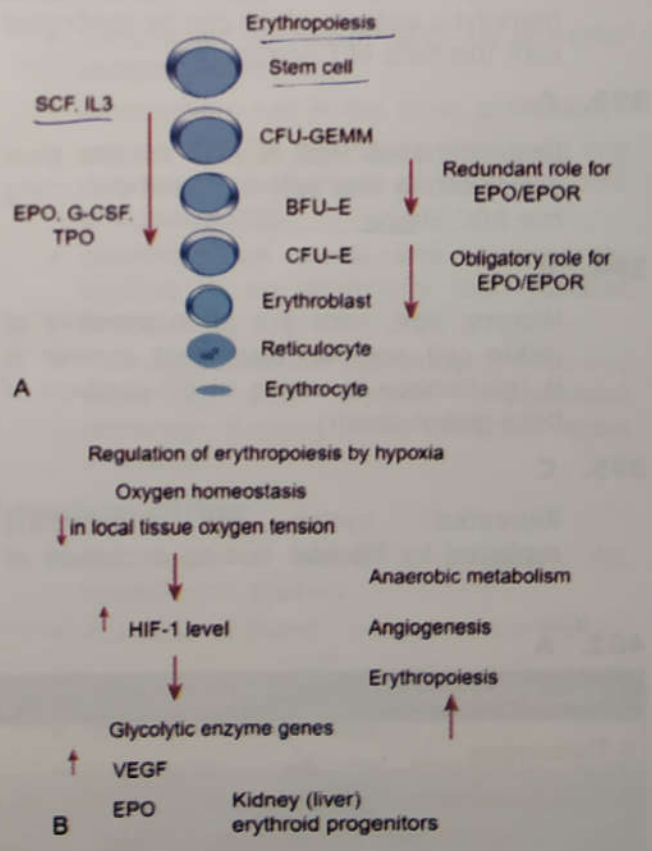
386. A

Fanconi anemia is the most frequently reported of the rare inherited bone marrow failure syndromes (IBMFSs), with approximately 2000 cases reported in the medical literature. In 1927, Guido Fanconi first reported 3 brothers with macrocytosis, pancytopenia, and physical abnormalities. Subsequent cases were clinically diagnosed because of the combination of aplastic and various characteristic physical anomalies. The vast majority (75%) of individuals with Fanconi anemia have at least one physical anomaly. The most common are short stature and cutaneous, skeletal, craniofacial, and genitourinary anomalies. Additionally, approximately 5% of patients with Fanconi anemia have at least 3 of the defining features of VATER, or VACTERL, association (vertebral anomalies, anal atresia, cardiovascular anomalies, tracheoesophageal fistula, renal and/or radial anomalies, limb defects). Furthermore, individuals with an expanded phenotype VACTERC-H (the highest incidence in the *FANCD1/BRCA2* genotype), regardless of hematologic status, must be evaluated for Fanconi anemia.

387. C

Dry tap means no yield on marrow aspirate. It is characteristically seen when hematopoietic cells in marrow are replaced by fat or by fibrosis.

388. A



389. C

Option A- AIHA can be both inside the vessel (intravascular) and in the spleen (extravascular)

Option B- Intravascular- physical damage

Option D- Intravascular

390. B

Most abundant cytoskeletal protein in the RBC

Responsible for the biconcavity

Deficiency seen in hereditary spherocytosis and elliptocytosis

391. C Dehydration causes increased MCHC along with the shape change.

Option A- Most common cause of HS

Option B- MCV may be normal or decreased

Option D- Hemolytic anemia causes compensatory erythropoiesis thus raised reticulocyte count

392. C

Adult age group, short history of anemia and spherocytes suggest autoimmune hemolytic anemia which can be confirmed with the help of Coombs test.

393. D

Deoxygenated HbS is less soluble thus precipitating into polymers and distorting the RBC shape.

394. B

History, age, race are all suggestive of sickle cell anemia, thus best answer is B (glutamate to valine at 6th position of beta globin chain)

395. C

Repeated cycles of infarctions replaced by fibrosis due to occlusion of microvasculature can be seen in sickle cell anemia.

396. B

In HbSC red cells the percentage of HbS is 50%, as compared with only 40% in HbAS cells. Moreover, HbSC cells tend to lose salt and water and become dehydrated, which increases the intracellular concentration of HbS. Both of these factors increase the tendency for HbS to polymerize. As a result, individuals who are compound heterozygotes for HbS and HbC have a symptomatic sickling disorder (termed HbSC disease), but it is milder than sickle cell disease

397. C

Deficiency of alpha leads to excess of beta which tetramerises and precipitates, thus leading to hemolysis.

398. C

Decreased life span of RBCs is responsible for protection against malaria.

399. A

Only hemolytic anemia with acquired genetic defect is PNH

400. A

PNH is a stem cell defect which can lead to aplastic anemia and hence a low LAP score is seen.

401. 38 B

IgG is warm hemolysin antibody as well as cold hemolysin antibody (Donath Landsteiner antibody)

402. A

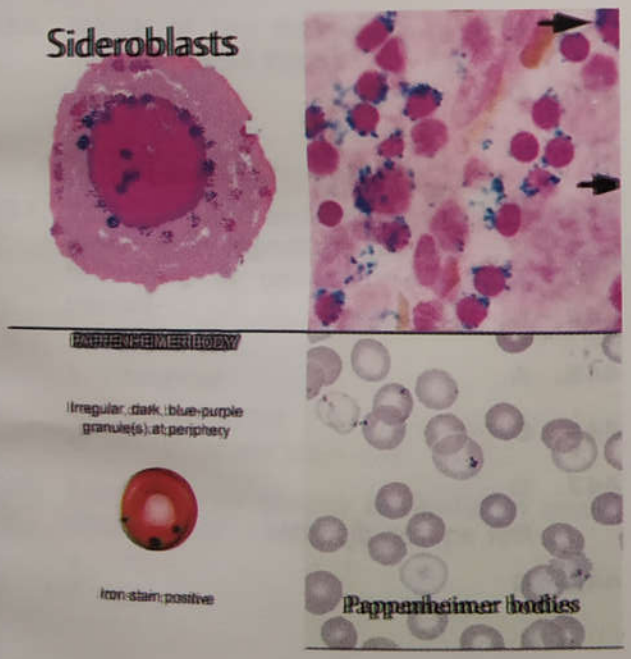
Clinical Syndromes	Genotype	Clinical Features	Molecular Genetics
β-Thalassemias			
β-Thalassemia major	Homozygous β-thalassemia (β^0/β^0, β^+/β^+, β^0/β^+)	Severe; requires blood transfusions	Mainly point mutations that lead to defects in the transcription, splicing, or translation of β-globin mRNA
β-Thalassemia intermedia	Variable (β^0/β^+, β^+/β^+, β^0/β, β^+/β)	Severe but does not require regular blood transfusions	
β-Thalassemia minor	Heterozygous β-thalassemia (β^0/β, $\beta+/\beta$)	Asymptomatic with mild or absent anemia; red cell abnormalities seen	

α-Thalassemias			
Silent carrier α-Thalassemia trait HbH disease Hydrops fetalis	–/ α/α –/ α/α (Asian) –/α –/α (black African, Asian) –/– –/ α –/– –/–	Asymptomatic; no red cell abnormality Asymptomatic, like β-thalassemia minor Asymptomatic, Asymptomatic, like β-thalassemia minor Severe; resembles β-thalassemia intermedia Lethal in utero without transfusions	Mainly gene deletions

403. D

Siderocytes are anucleate erythrocytes with iron-containing (siderotic) cytoplasmic inclusions. The inclusions can be due to aggregates of iron in the cytoplasm or within mitochondria (the latter are called Pappenheimer bodies). Can be visualised on both Romanowsky and Perl's stain.

A nucleated erythoid cell with the same inclusions (usually within mitochondria) is termed a sideroblast which can be visualised in marrow only on Perl's stain.



404. A

Option B- most characteristic finding

Option D- remnants of mitotic spindles (microtubules)

405. A

EDTA and sodium citrate remove calcium which is essential for coagulation.

Calcium is either precipitated as insoluble oxalate or bound in non ionized form

Heparin binds to anti thrombin thus inhibiting interaction of several clotting factors

EDTA is used for blood counts

Sodium citrate is used for coagulation testing and ESR

Any anticoagulant can be used for collecting blood for flow cytometry

EDTA

- sodium and potassium salts are powerful anticoagulants
- dipotassium salt is the most preferable
- excess of EDTA can cause shrinkage and degenerative changes of both red cells and leukocytes
- platelets can swell and disintegrate leading to an artificially high platelet count
- leucoagglutination can occur (naturally occurring anti platelet antiplatelet antibody- if seen, blood collected again in citrate)

Trisodium citrate

- 9:1 ratio of blood : coagulant for coagulation studies
- 4:1 ratio of blood : coagulant for ESR

Heparin

- lithium or sodium salt
- commonly used for chemistry, gas analysis and emergency tests
- does not alter size of red cells
- best used for OFT
- not suitable for blood counts as it induces platelet and leucocyte clumping
- not used to make smears as it gives a blue discoloration with Romanowsky dyes

- not used in PCR as it inhibit enzyme activity

406. C

Fibrosis is caused by tumor cells

407. A

Anisocytosis is a feature of IDA, not beta thalassemia trait

408. D

Malabsorption → megaloblastic anemia

409. C

Both are .46 microcytic hypochromic

410. C

Parvovirus B19 infection causes aplastic crisis

411. C

Autosomal dominant is most common inheritance

412. A

Acquired, not an inherited genetic trait

413. C

414. B

415. C

416. A

417. A

418. A

419. C

420. C

421. C

422. A

423. A

424. A

425. C

426. A

427. A

428. C

429. A

430. D

431. A

432. A

433. A

434. B

435. B

436. D

437. B

438. B

439. B

440. D

441. B

442. A

443. D

444. D

445. A

446. D

447. D

Not an iron disorder

448. D

WBC Disorders – Questions

449. Dohle bodes with giant platelets are seen in?

a. May heggelin anomaly
b. Pelger huet anomaly
c. Chediak higashi syndrome
d. Bernard Soulier syndrome

450. Marker of T-lymphocyte is?

a. CD8
b. CD20
c. CD19
d. CD45

451. Eosinophillia is found in?

a. Cryptococcus
b. HPV
c. Stronglyloides
d. Typhoid

452. Dilated endoplasmic reticulum is called as?

a. Asteroid bodies
b. Ferruginous bodies
c. Hirano bodies
d. Dohle bodies

453. Which of the following is a B cell marker? (PGI type)

a. CD22
b. CD10
c. CD1a
d. CD19
e. CD20

454. In an ablated animal, myeloid series cells are injected. Which of the following is seen after incubation period-

a. RBC
b. Fibroblast
c. T lymphocytes
d. Hematopoetic stem cell

455. Bone marrow biopsy is useful in the diagnosis of- (PGI)

a. CML
b. ALL
c. Aleukemic leukemia
d. Hodgkins disease
e. ITP

456. A patient presented with painless b/l proptosis. What is the next investigation to diagnose it as chloroma?

a. Blood haemoglobin
b. Peripheral smear
c. Platelets
d. Bone marrow (reticulin)

457. All of the following are seen in the development of T lymphocyte at a point except?

a. Tdt
b. CD34
c. PAX5
d. CD1a

458. Good prognosis for ALL is?

a. Age > 10 years
b. Hyperdiploidy
c. T cell variant
d. TLC > 1,00,000/ul

459. Most common translocation in acute promyelocytic leukemia? (PGI)

a. t(8:14)
b. t(15:17)
c. t(9:22)
d. t(8:11)
e. t(11:14)

460. Which one is the best prognostic factor for ALL?

a. Hyperploidy
b. Organomegaly
c. TLC more than 50,000/ul
d. Response to treatment

461. Stain used for diagnosis of granulocytic sarcoma:

a. Myeloperoxidase
b. Leukocyte alkaline phosphatase
c. Nonspecific esterase
d. Neuron specific enolase

449. a	450. a	451. c	452. d	453. a,b,d,e	454. a	455. b,c	456. b
457. c	458. b	459. b	460. d,a	461. a			

462. Most common type of AML in Down's syndrome:

a. M2 b. M3
c. M6 d. M7

463. A 17-years-old boy presented with TLC of 138x 109/L with 80% blasts on the peripheral smear. Chest X-ray demonstrated a large mediastinal mass. Immunophenotyping of this patient's blasts would most likely demonstrate-

a. No surface antigens (null phenotype)
b. An immature T cell phenotype (Tdt CD34/CD7 positive)
c. Myeloid markers, such as CD13, CD33 and CD15
d. B cell markers, such as CD19, CD20 and CD22

464. A 15-years-old boy presented with one day history of bleeding gums, sub-conjunctival bleed and purpuric rash. Investigations revealed the following results: Hb-6.4 gm/dL; TLC-26,500/mm3, Platelet 35,000/mm3; prothrombin time-20 sec with a control of 13 sec; partial thromboplastin time-50 sec; and Fibrinogen 10mg/dL. Peripheral smear was suggestive of Acute Myeloblastic Leukemia. Which of the following is the most likely-

a. Myeloblastic leukemia without maturation
b. Myeloblastic leukemia with maturation
c. Promyelocytic leukemia
d. Myelomonocytic leukemia

465. Lymphohistocytic variant of Reed Sternberg seen in which subtype of Hodgkin lymphoma?

a. Nodular sclerosis
b. Lymphocyte rich
c. Lymphocyte predominant
d. Lymphocyte depleted

466. A 20-years-old male presented with cervical lymphadenopathy. Histology of lymph node shows RS cell with vague nodule formation and background T reactive lymphocytes. The cells were positive for CD20, LCA, EMA and negative for CD 15 and CD30. Diagnosis is?

a. NLPHL
b. T cell rich B cell lymphoma
c. Nodular sclerosis Hodgkin
d. CLL

467. EBV is not associated with?

a. Lymphocyte predominant HD
b. Plasmablastic lymphoma
c. Nasopharyngeal Ca
d. Mixed cellularity HD

468. Flow cytometry is done on:

a. Polycythemia
b. Thrombocytosis
c. Basophilia
d. Lymphocytosis

469. CLL/SLL ARISES FROM WHICH CELL?

a. Mature B cell
b. Naïve B cell
c. Centrocytes of germinal center
d. Progenitor B-cell

470. Which of the following immunohisto chemistry marker is used in Cyclin D1 negative Mantle cell lymphoma?

a. SOX11 b. ITRA1
c. MYD88 d. Annexin V

471. A 35-years-old presented with fever. On examination he had enlarged and ulcerated tonsils. His peripheral blood smear showed lymphocytosis. Monospot test was negative. Tonsillectomy was done. The biopsy of the same showed large cells mixed with lymphocytes. The cells were positive for CD20, EBVLMP1, MUM1, CD79a. Background cells were positive for CD3. The cells are negative for CD15. Your most probable diagnosis?

a. Infectious mono-nucleosis
b. Hodgkin lymphoma
c. EBV positive – DLBL
d. EBV positive mucocutaneous ulcer

462. d	463. b	464. c	465. c	466. a	467. a	468. d	469. b	470. a	471. c

472. PAX-5 is a marker for (PGI type)
a. Diffuse large B-celllymphoma
b. Hodgkins lymphoma
c. Anaplastic lymphoma
d. Lymphoblastic lymphoma
e. AML

473. TRAP positivity is seen in?
a. Hairy cell leukemia
b. ALL
c. Burkitts lymphoma
d. T-cell leukemia

474. Cyclin D1 is expressed in-
a. Follicular lymphoma
b. Chronic lymphoid lymphoma
c. Mantle cell lymphoma
d. Splenic marginal zone lymphoma

475. CD20 is positive in all the following lymphomas except:
a. Mantle cell lymphoma
b. Lymphocyte rich HL
c. Follicular lymphoma
d. Butkitt lymphoma

476. A 60-years-old male living in hilly area has Hb of 16 gm%, TC 21000/ul. DLC showed metamyelocytes and myelocytes 40%, N25% L40%, E5%. Platelet count 3.25 lakh/u. He presented with hypertension and on examination, spleen was just palpable below costal margin. What is the next step?
a. Bone marrow with reticulin stain
b. JAK STAT mutation assessment
c. Philadelphia chromosome
d. Erythropoietin levels

477. 45/m present with leuko-erythro-blastic blood picture with dacrocytes. What is bone marrow finding?
a. Fatty degeneration with erythroid cell hyperplasia with megakaryocytes
b. Abundant fibrosis
c. Focal cellular marrow with hypocellular areas and atypical megakaryocytes.
d. Hypercellular marrow with prominent blasts

478. Mutation seen in systemic masto-cytosis:
a. FGFR1 fusion genes
b. BCR-ABL fusion gene
c. JAK 2 point mutation
d. c-kit point mutation

479. A 60-years-old man presented with fatigue, weight loss and heaviness in left hypochondrium for 6 months. The hemo-gram showed Hb=10 gm/dL, TLC 5 lakhs/mm3, platelet count 4 lakhs/mm3, DLC neutrophil 55% lymphocytes 4% monocytes 2% basophils 6% metamyelocytes 10%, myelocytes 18%, promyelocytes 2% and blasts 3%. The most likely cytogenetic abnormality in this case is:
a. t(1;21)
b. t(9;22)
c. t(15;17)
d. Trisomy 21

480. A 50-years-old man presents with fatigue and easy bruising. A complete blood count and peripheral blood smear show leucocytosis with 56% circulating blasts. The patient has no previous history of myeloid neoplasm or treatment with chemotherapy. Which of the following findings would definitively establish a diagnosis of acute myeloid leukemia?
a. Presence of blasts with deeply basophilic and vacuolated cytoplasm
b. Presence of Auer rods in the blasts
c. Presence of small blasts with round nuclei, variably dispersed chromatin, occasional nucleoli and scant amounts of basophilic cytoplasm
d. Presence of large blasts with round nuclei, dispersed chromatin, prominent nucleoli and abundant basophilic cytoplasm

481. Detection of which of the following molecular genetic abnormalities is equivalent to a diagnosis of acute myeloid leukemia, regardless of blast count?
a. t(8;21)(q22;q22)
b. t(9;11)(q22;q23)
c. t(6;9)(q23;q34)
d. t(3;3)(q21;q26.2)

472. a,b,d	473. a	474. c	475. b	476. b	477. b	478. d	479. b	480. b
481. a								

482. Which of the following molecular genetic abnormalities results in the disruption of core binding factor-α?

a. Inv(16)(p13.1q22)
b. t(16;16)(p13;1;q22)
c. t(6;9)(p23;q34)
d. t(8;21)(q22;q22)

483. Which of the following statements regarding acute promyelocytic leukemia with PML-RARA is the most correct?

a. The disease shows a poor response to treatment
b. The disease is almost always preceded by chronic myelogenous leukemia
c. Patients are at high risk for the development of disseminated intravascular coagulation
d. Most patients present in the seventh or eight decade of life

484. Which of the following findings would not be expected in the blast population of acute myeloid leukemia with monocytic differentiation?

a. Expression of CD14
b. Expression of CD34
c. Expression of CD64
d. Expression of CD4

485. Which of the following molecular genetic abnormalities would be most likely to impart a poor prognosis in a patient with acute myeloid leukemia?

a. T(8;21)(q22;q22); RUNX1-RUNX1T1
b. Normal karyotype
c. Complex karyotype
d. T(15;17)(q22;q12); PML-RARA

486. A 70-years-old man presents with pancytopenia for six months. A bone marrow is performed and shows dysplasia in the granulocyte, erythrocyte and megakaryocyte lineages. The blast count in the peripheral blood is less than 1% and the blast count in the bone marrow is less than 5%. The best diagnosis is:

a. Myelodysplastic syndrome with excess blasts-1
b. Myelodysplastic syndrome with excess blasts-2
c. Myelodysplastic syndrome with multilineage dysplasia
d. Acute myeloid leukemia with myelodysplasia-related changes

487. All of the following are features of accelerated phase of chronic myeloid leukemia, BCR-ABL1 positive, except:

a. Presence of a second Philadelphia chromosome
b. Presence of 20% blasts in the peripheral blood
c. Presence of 20% basophils in the peripheral blood
d. Persistent thrombocytosis (>1000 x 109/L), unresponsive to therapy

488. Follicular lymphoma is characterized by the following cytogenetic abnormality:

a. t(8;14)
b. t(14;18)
c. t(15;17)
d. t(11;14)

489. A characteristic feature of lymph node involved by CLL/SLL is:

a. Effacement of lymph node architecture with presence of large and small cells
b. Presence of pseudofollicles
c. Marked increase in vascularity
d. Large cells resembling Reed-Sternberg cells

490. Thesaurocytes are:

a. Plasma cells with flame shaped cytoplasm
b. Plasma cells with ground glass cytoplasm
c. Plasma cell with intra nuclear inclusion
d. Binucleated plasma cells

482. d	483. c	484. d	485. c	486. a	487. b	488. b	489. b	490. a

491. Which of the following malignancy is associated with underlying progression and spreads characteristically in a stepwise fashion and hence staging the disease is an important prognostic factor?

a. Hodgkin's lymphoma
b. Multiple myeloma
c. Mature T cell NHL
d. Mature B cell NHL

492. International Prognostic index for lymphomas includes the following prognostic factors, except

a. Stage of disease
b. Number of extralymphatic sites involved
c. LDH
d. Hemoglobin and albumin

493. A 48-years-old woman was admitted with a history of weakness for two months. On Examination, cervical lymph nodes were found enlarged and spleen was palpable 2 cm below the costal margin. Her haemoglobin was 10.5 g/dL, platelet count 2.7 x 109/L and total leukocyte count 40 x 109/L, which included 80% mature lymphoid cells with coarse clumped chromatin. Bone marrow revealed a nodular lymphoid infiltrate. The peripheral blood lymphoid cells were positive for CD19, CD5, CD20 and CD23 and were negative for CD79 B and FMC-7. The Histopathological Examination of the lymph node in this patient will most likely exhibit effacement of lymph node architecture by-

a. A pseudofollicular pattern with proliferation centers
b. A monomorphic lymphoid proliferation with a nodular pattern
c. A predominantly follicular pattern
d. A diffuse proliferation of medium to large lymphoid cells with high mitotic rate

494. Burkitt's lymphoma is positive for-

a. CD5
b. CD15
c. CD20
d. CD25

495. Most common extranodal site of lymphoma in HIV is?

a. CNS
b. GIT
c. Retroperitoneum
d. Mediastinum

496. Post-transplant lymphoma occurs due to proliferation of which of the following cells-

a. T cell
b. B cell
c. NK cell
d. Monocyte

497. DIC is common in which AML-

a. Monocytic (M5)
b. Promyelocystic (M3)
c. Erythroblastic (M6)
d. Megakaryocytic (M7)

498. A 10 years old child presents with pallor and history of blood transfusion 2 months back. On investigation, Hb 4.5 gm, total count 60000, platelet count 2 lakh and CD10 (+) ve, CD19 (+) ve, CD117 (+) ve, MPO (+) ve& CD33(-) ve. What is the most likely diagnosis?

a. ALL
b. AML
c. Undifferentiated leukemia
d. Mixed phenotypic acute leukemia

499. Compared to the other leukemias, hairy cell leukemia is associated with which of the following infections-

a. Parvovirus B19
b. Mycoplasma
c. Atypical mycobacteria
d. Salmonella

500. Mycosis fungoides is-

a. T cell ymphoma
b. B cell ymphoma
c. Mixed
d. Plasma cell tumour

501. Which of the following M protein immunoglobulin is not synthesized in multiple myeloma?

a. IgD
b. IgM
c. IgA
d. IgG

491. a	492. d	493. a	494. c	495. a	496. b	497. b	498. d	499. c	500. a
501. b									

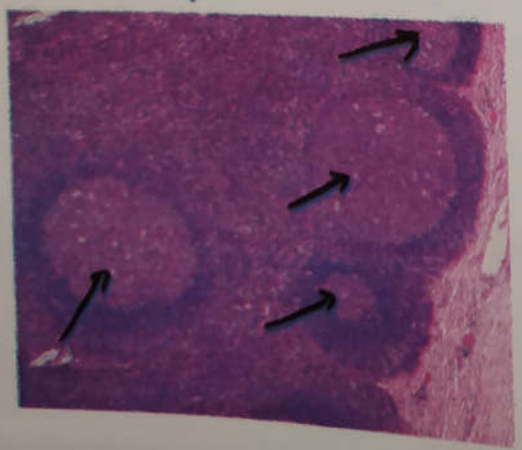
502. Russel bodies are seen in-
a. Lymphocytes
b. Neutrophils
c. Macrophage
d. Plasma cells

503. Not true regarding Waldenstorm's macroglobulinemia is-
a. Lymphadenopathy is usually present
b. Blood viscosity increased
c. IgM immunoglobulin increased
d. Hypercalcemia

504. Lethal midline granuloma arises from-
a. T cells
b. B cells
c. NK cells
d. Macrophages

505. A patient with increased TLC was investigated for leukemia. On flow cytometry the following profile was obtained.
CD 34 positive
CD 11c positive
CD 14 positive
CD 64 positive
CD 22 positive
CD 19 positive
CD 10 positive
The diagnosis is?
a. Acute lymphoblastic leukemia with aberrant myeloid expression
b. Acute monoblastic leukemia with aberrant lymphoid expression
c. Mixed phenotypic acute leukemia
d. Acute myeloid leukemia

506. The labelled area in the lymph node can be highlighted by which of the following markers on immuno-histochemistry



a. CD 3
b. CD 20
c. CD 56
d. CD 68

507. A child presents with protrusion of eyes, increased thirst and diuresis and bony defects in the calvarium. A biopsy was performed and electron microscopy showed the following. Which marker would confirm the diagnosis of this condition?

a. S-100
b. MHC II
c. CD 207
d. All of the above

508. All of the following stem cell populations are found within the bone marrow, except-
a. Endothelial Progenitor cells
b. Myoblast Progenitor cells
c. Mesenchymal stem cells
d. Hematopoietic stem cells

509. Leukocyte common antigen is:
a. CD45
b. CD20
c. CD19
d. CD41

510. CD30 is/are marker for (PGI Type)
a. Anaplastic large cell lymphoma
b. Embryonal carcinoma
c. Squamous cell carcinoma
d. Seminoma
e. Hodgkin's lymphoma

511. Bimodal distribution is seen in?
a. Hodgkins lymphoma
b. DLBCL
c. ALL
d. CML

502. d	506. d	504. c	505. c	506. b	507. d	508. b	509. a	510. a,b,e	511. a

512. Primary extranodal neoplasms is/ are (PGI type)

a. Burkitt lymphoma
b. Waldeyer's ring lymphoma
c. Gastric lymphoma
d. Thyroid lymphoma
e. Mycosis fungoides

513. Which of the following is not a characteristic feature of Myelodysplastic syndrome?

a. Leucoerythroblastic blood picture
b. Pawn ball megakaryocytes
c. Pseudo pelger heut cells
d. Transformation to AML

514. The following is true regarding polycythemiavera?

a. Raised ESR
b. Decreased LAP score
c. Thrombocytopenia
d. Leukocytosis

515. Triad of leukoerythroblastosis, tear drop erythrocytes and large platelets is seen in:

a. Essential thrombocytosis
b. Primary myelofibrosis
c. Myelodysplastic syndrome
d. Langerhan cell histiocytosis

516. Which of the following conditions is most likely to lead to a dry tap during bone marrow aspiration?

a. Acute lymphoblastic leukemia
b. Multiple myeloma
c. Megaloblastic anemia
d. Idiopathic myelofibrosis

517. CD marker for Langerhans cellhistiocytosis is?

a. CD 17 b. CD 23
c. CD 1a d. CD117

518. Which of the following findings would be most useful for establishing myeloid lineage in acute leukemia?

a. Expression of CD13 by the blasts
b. Expression of CD33 by the blasts
c. Expression of myeloperoxidase by the blasts
d. Expression of cytoplasmic CD3 by the blasts

519. Which of the following antigens is not typically expressed in acute promyelocytic leukemia with PML-RARA?

a. CD13 b. CD33
c. MPO d. HLA-DR

520. On which chromosome is the retinoic acid receptor α gene located?

a. 15q22 b. 17q12
c. 18q11.2 d. 11q13

521. Which of the following is the strongest predictor of response to therapy in acute myeloid leukemia?

a. Percentage of blasts in the peripheral blood
b. Immunophenotype of the leukemic blasts
c. Percentage of blasts in the bone marrow
d. Karyotype of the leukemic blasts

522. All of the following are causes of blasts in the peripheral blood except:

a. Myelodysplastic syndrome
b. Chronic myeloid leukemia, BCR-ABL1+
c. Chronic lymphocytic leukemia
d. Sepsis

523. Which of the following is an unfavourable factor for a child with B-lymphoblastic leukemia/ lymphoma?

a. Being six years of age
b. Presence of t(12;21) (p13.2;q22.1);ETV6-RNX1
c. Presence of MLL (KMT2A) gene rearrangement
d. Lack of central nervous system involvement at presentation

524. Which of the following antigens is specific for B-lymphoblastic leukemia/lymphoma by flow cytometry?

a. CD19 b. CD10
c. TdT d. None of the above

512. a,b,c,d,e	513. a	514. d	515. b	516. d	517. c	518. c	519. d	520. b
521. d	522. c	523. c	524. a					

525. Which of the following is not a feature of myelodysplastic syndrome with ringed sideroblasts?

a. >15% ring sideroblasts (as a percentage of all erythroid cells) in the bone marrow
b. Anemia
c. 2% blasts with Auer rods in the peripheral blood
d. Presence of SF3B1 mutation

526. Which of the following statements regarding t(9;22)(q34;q11.2); BCR-ABL1 and its resultant fusion proteins is false?

a. The fusion protein p210 is most frequently seen in chronic myeloid leukemia, BCR-ABL1 positive
b. The fusion protein p190 is most frequently seen in Philadelphia chromosome-positive B-lymphoblastic leukemia/lymphoma
c. The fusion protein p230 is associated with morphologic features that resemble chronic myelomonocytic leukemia.
d. The fusion protein p230 is associated with increased neutrophilic maturation in chronic myeloid leukemia

527. The highest incidence of JAK2 mutation is seen in:

a. Polycythemiavera
b. Primary myelofibrosis
c. Essential thrombocythemia
d. Chronic myeloid leukemia, BCR-ABL1 positive

528. All of the following abnormalities may be seen in primary myelofibrosis except:

a. JAK2 mutation
b. BCR-ABL1 fusion
c. CALR mutation
d. MPL mutation

529. Neoplastic mast cells are positive for;

a. CD20
b. CD3
c. CD25
d. CD117

530. Detection of which of the following antigens by flow cytometry is diagnostic of T-cell differentiation in acute lymphoblastic leukemia?

a. CD1a
b. Cytoplasmic CD22
c. Cytoplasmic CD3
d. Surface CD4

531. The follicles in a case of follicular lymphoma are expected to be positive for:

a. CD10
b. Bcl 6
c. Bcl 2
d. All of the above

532. All of the following regarding chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL) are true except:

a. CLL/SLL is the most common leukemia in Western countries
b. Radiation exposure does not increase the risk of developing CLL/SLL
c. Most patients at presentation have fever and weight loss
d. CLL/SLL patients are typically over 50 years of age

533. Which of the following imparts a better prognosis for patients with CLL/SLL?

a. CD38 negativity
b. ZAP-70 positivity
c. Presence of Trisomy 12
d. Lymphocyte doubling time of 8 months

534. Mantle cell lymphoma should be positive for all of the following markers except:

a. Cyclin D1
b. CD 20
c. CD 10
d. CD 5

535. Which of the following is not a risk factor for the development of extranodal marginal zone lymphoma (MALT lymphoma)?

a. Sjogren's syndrome
b. Pernicious anemia
c. Helicobacter pylori infection
d. Hashimoto's thyroiditis

525. c	526. c	527. a	528. b	529. c	530. c	531. d	532. b	533. a	534. c
535. b									

536. The following are all features of nodal marginal zone lymphoma except:

a. Tumor cells are CD79a positive
b. Tumor cells are Bcl2 positive
c. Tumor cells are CD10 negative
d. Tumor cells are Bcl6 negative

537. All of the following are characteristic histologic features of Burkitt lymphoma except:

a. Diffuse growth pattern
b. Starry sky appearance
c. Large cells with inconspicuous nucleoli
d. High mitotic rate

538. Which of the following positive immune histochemical stains should make one reconsider a diagnosis of Burkitt lymphoma?

a. CD10 positivity
b. Bcl2 positivity
c. Bcl 6 positivity
d. CD20 positivity

539. Which of the following translocations may be seen in Burkitt lymphoma?

a. t(8;14)
b. t(2;8)
c. t(8;22)
d. All of the above

540. Which of the following is a true statement regarding lympho-plasmacytic lymphoma (LPL)?

a. It is synonymous with Waldenstrom macroglobulinemia
b. This is a high-grade lymphoma
c. It is associated with hepatitis C infection
d. It is not known to transform to a high-grade lymphoma

541. Which of the following variants of diffuse large B-cell lymphoma (DLBCL) is not associated with EBV infection?

a. DLBCL associated with chronic inflammation
b. Intravascular large B-cell lymphoma
c. Primary effusion lymphoma
d. Lymphomatoid granulomatosis

542. Primary effusion lymphoma patients may be infected with:

a. HIV
b. EBV
c. HHV-8
d. All of the above

543. All of the following are features of hairy cell leukemia except:

a. The disease involves the red pulp of the spleen
b. There is leucocytosis
c. Monocytopenia occurs
d. Bone marrow aspiration may result in a dry tap

544. All of the following markers are expected to be positive in hairy cell leukemia except:

a. Annexin 1
b. CD25
c. CD103
d. CD3

545. Which of the following neoplastic cell types is not expected to be seen in a case of classical Hodgkin lymphoma?

a. Reed-Sternberg cell
b. L&H cell
c. Lacunar cell
d. Mummified cell

546. Which of the following is not a subtype of classical Hodgkin lymphoma?

a. Nodular sclerosis
b. Eosinophil rich
c. Lymphocyte depleted
d. Lymphocyte rich

536. b	537. c	538. b	539. d	540. a	541. a	542. d	543. d	544. d
545. b	546. b							

547. A 55-years-old Caucasian woman presents for evaluation of fatigue. The patient states that her symptoms have been present for approximately eight months and reports two episodes of cellulitis over the last year. Physical examination reveals mild splenomegaly; no skin lesion, lymphadenopathy, or other abnormalities are identified. Review of a complete blood count (CBC) and peripheral blood smear shows absolute neutropenia, normocytic, normochromic anemia, and mild absolute lymphocytosis composed of numerous large lymphocytes with abundant cytoplasm and prominent red-pink granules. Which of the following is the most likely diagnosis?

a. T-cell prolymphocytic leukemia
b. T-cell large granularlym-phocyticleukemia
c. Adult T-cell leukemia/lymphoma
d. Aggressive NK-cell leukemia

548. Classical markers for Hodgkin's disease

a. CD15 and CD30
b. CD15 and CD22
c. CD15 and CD20
d. CD20 and CD30

549. Lacunar cells are seen in which type of Hodgkin's lymphoma-

a. Lymphocyte predominant
b. Lymphocyte depletion
c. Nodular sclerosing
d. Mixed cellularity

550. 'Popcorn-cells' are seen in which variety of Hodgkin's disease-

a. Nodular sclerosis
b. Mixed cellularity
c. Lymphocyte predominant
d. Lymphocyte depletion

551. All of the following are true about nodular sclerosis of Hodgkin's disease except-

a. Well-formed collagen bands
b. CD15+
c. CD20+
d. Infiltration by plasma cells

552. Which cell is/are in not seen in Hodgkin's lymphoma?

a. Reed-Sternberg cell
b. Lacunar cell
c. L & H cell
d. Langerhans cell
e. Hodgkin's cell

553. A 63 years-old man presented with massive splenomegaly, lymphade-nopathy and a total leucocyte count of 17000 per mm1. The flow cytometry showed CD23 negative and CD5 positive monoclonal B cells with bright kappa positively comprising 80% of the peripheral blood lymphoid cells. The most likely diagnosis is-

a. Mantle cell lymphoma
b. Splenic lymphoma with villous lymphocytes
c. Follicular lymphoma
d. Hairy cell leukemia

554. Starry sky appearance is seen in-

a. Burkitt's lymphoma
b. CLL
c. Diffuse large B cell lymphoma
d. ALCL

555. Maltoma is positive for-

a. CD3 b. CD10
c. CD43 d. CD5

556. Poor prognostic indicator of All is-

a. Female sex
b. Leukocyte count < 50,000
c. Age greater than 1 year
d. Hypodiploidy

557. t (2;8) is characteristically seen with-

a. Pre B cell lymphoma
b. Pre T cell lymphoma
c. Burkitt's lymphoma
d. Mantlecelllymphoma

558. Most common extranodal site for Non-Hodgkin's lymphoma is-

a. Stomach b. Brain
c. Intestines d. Tonsils

547. c	548. a	549. c	550. c	551. c	552. d	553. a	554. a	555. d	556. d
557. c	558. a							

559. B ALL is due to-

a. T cells
b. Immature B cells
c. Immature T cells
d. Both T and B cells

560. CD10 is seen in-

a. ALL
b. CLL
c. HCL
d. CML

561. Good prognosis of ALL

a. Hyperdiploidy
b. Hypodiploidy
c. T cell lineage
d. Philadelphia chromosome

562. All of the following are good prognostic factors for ALL except-

a. Age of onset between 2-8 years
b. Initial WBC count less than 50000
c. Hyperdiploidy
d. t (9:22), t (8:14), t (4:11)

563. Nonspecific esterase in present in-

a. Megakaryocytic leukemia
b. Lymphocytic leukemia
c. Erythroleukemia
d. AML M4 and M5

564. What is the chromosomal translocation in AML M3?

a. T (18,21)
b. T (15,17)
c. T (8,21)
d. T (9,11)

565. AML is characterized by-

a. Philadelphia chromosome
b. Auer rods
c. Hemolyticanemia
d. Dohle bodies

566. A 50 years old patient comes with pancytopenia, splenomegaly and skin lesion (erythema nodosum). Most probable diagnosis is:

a. Mycosis fungoides
b. Myledysplastic syndrome
c. Hairy cell leukemia
d. Sezary syndrome

567. Marker for hairy cell leukemia is-

a. CD30
b. CD103
c. CD1a
d. CD4

568. Hyperviscosity syndrome occurs with which of the following types of M protein of Multiple myeloma?

a. IgG1
b. IgG2
c. IgG3
d. IgG4

569. Most common cytogenetic abnormality in multiple myeloma?

a. Deletion 13q
b. t (11;14)
c. t (8;14)
d. t (8;22)

570. A 2 years old child presents with scattered lesions in the skull. Biopsy revealed Langerhans cells. The most commonly associated is marker with this condition will be-

a. CD1a
b. CD57
c. CD3
d. CD68

571. Birbeck granules in the cytoplasm are seen in-

a. Mast cells
b. Langerhans cells
c. Thrombocytes
d. Myelocytes

572. CD marker of histiocytosis is-

a. CD1a
b. CD1b
c. CD1c
d. CD1d

573. Dohle bodies-

a. Dilated rough endoplasmic reticulum in neutrophils
b. Mitochondria
c. Golgi apparatus
d. Lysosomes

574. Auer rods are seen maximally in-

a. M2-AML
b. M3-AML
c. M6-AML
d. ALL

575. BCL2 in lymphoma the translocation involving-

a. t (8:14)
b. t (8:12)
c. t (14:18)
d. t (14:22)

559. b	560. a	561. a	562. d	563. d	564. b	565. b	566. c	567. b	568. c
569. b	570. b	571. b	572. a	573. a	574. b	575. c			

WBC Disorders – Explanations

449. A

May-Hegglin anomaly (MHA) is an autosomal dominant disorder characterized by various degrees of thrombocytopenia that may be associated with purpura and bleeding; giant platelets containing few granules; and large, well-defined, basophilic, cytoplasmic inclusion bodies in granulocytes that resemble Döhle bodies.

Option B- bilobed neutrophils

Option C- large coarse granules with albinism and neuropathies

Option D- giant platelets

450. A

Option B and C- B cell markers

Option D- Leucocyte commonantigen (LCA)

451. C

Helminthic infections induce eosinophilia.

452. D

In sepsis or severe inflammatory disorders (e.g., Kawasaki disease), leukocytosis is often accompanied by morphologic changes in the neutrophils, such as toxic granulations, Döhle bodies, and cytoplasmic vacuoles. Toxic granules, which are coarser and darker than the normal neutrophilic granules, represent abnormal azurophilic (primary) granules. Döhle bodies are patches of dilated endoplasmic reticulum that appear as sky-blue cytoplasmic "puddles."

Option A- seen in sarcoidosis

Option B- iron rich proteinaceous material covered particles seen in pneumoconiosis

Option C-

453. A, B, D, E

Option C- thymocytes and Langerhans cells

454. A

Myeloid series cells include RBCs, platelets, monocytes and granulocytes.

455. B, C

Option A- Aspirate is mandatory for diagnosis as counting of cells needed

Option C- absence of tumor cells in blood mandate an investigation of the marrow to determine blast count

Option E- due to antibodies against platelet antigens, hence marrow will show megakaryocyte hyperplasia but not specific for diagnosis of ITP

456. B

Chloroma or myeloblastoma or granulocytic sarcoma is extramedullary blast proliferation which may coexist with acute myeloid leukemia and hence a primary first line investigation of the peripheral blood is needed.

Option A- Establishing anemia will not contribute to diagnosis

Option C- No history of bleeding- hence not needed here

Option D- to determine fibrosis in marrow- not needed here

457. C

PAX5 is a B cell transcription factor and has no role in T cell development

458. B

Prognostic Factors in Acute Lymphoblastic Leukemia		
Determinants	Favorable	Unfavorable
White blood cell counts	$<10 \times 109/L$	$>200 \times 109/L$
Age	3–7 y	<1 y, >10 y

Gender	Female	Male
Ethnicity	White	Black
Node, liver, spleen enlargement	Absent	Massive
Testicular enlargement	Absent	Present
Central nervous system leukemia	Absent	Overt (blasts + pleocytosis)
FAB morphologic features	L1	L2
Ploidy	Hyperdiploidy	Hypodiploidy <45
Cytogenetic markers	Trisomies 4, 10, and/or 17	t(9;22) (BCR-ABL)
	t(12;21) (TEL-AML1)	t(4;11) (MLL-AF4)
Time to remission	<14 d	>28 d
Minimal residual disease	<10-4	>10-3

Prognostic Factors For Remission Duration in Adults with Acute Lymphoblastic Leukemia (All)	
Patient Features	Prognostic Factor
Age (y)	
<30	Favorable
30	Unfavorable
White blood cell count ×(106/ml)	
<30,000	Favorable
30,000 (>100,000 for T cell)	Unfavorable
Immunophenotype	
T-cell ALL	Favorable
Mature B-cell ALL; early T-cell ALL	Unfavorable
Cytogenetics	
12p abnormality; t(10;14)(q24;q11)	Favorable
Normal; hyperdiploid	Intermediate
t(9;22), t(4;11), t(1;19), hypodiploid, –7, +8	Unfavorable
Response to therapy	
Complete remission within 4 wk	Favorable
Persistent minimal residual disease	Unfavorable

459. B

Option A- Burkitt lymphoma

Option C- CML, ALL AML

Option E- Mantle cell lymphoma, Plasma cell Myeloma

460. D, A

In order of preference, response of treatment, cytogenetics and clinical features are considered to be important prognostic factors for ALL.

461. A

The MPO reaction is positive in cells of the granulocytic series and may be positive in monocytes. The basis of the stain is breakdown of hydrogen peroxide by the enzyme MPO, releasing an oxygen radical that reacts with a soluble substrate (e.g., diaminobenzidine or o-tolidine) to form a colored precipitate. MPO is located in the peroxisomes of neutrophils and monocytes and specific granules of eosinophils.

Lymphoid cells and erythroid precursors are negative.

SBB is a direct stain of phospholipid in granule membranes. The pattern of staining closely parallels that of MPO, but the SBB reaction is usually stronger. Monoblasts may contain sudanophilic granules. Erythroid precursors are negative.

Auer rods are MPO- and SBB-positive.

Nonspecific esterase reactivity is found

in monocytes. The basis for the esterase cytochemical stains is enzymatic release of a side chain from a naphthol ring, with subsequent reaction of the free ring with a soluble color developer to generate a colored precipitate. The most commonly used substrates for NSE are α-naphthyl butyrate (ANB) and α-naphthyl acetate (ANA).

Megakaryoblasts are negative with ANB staining. These features may be used to identify megakaryoblasts

The PAS stain reacts primarily with glycogen, generating a fuchsia-colored precipitate. Lymphoblasts in ALL often have prominent coarse granular or block PAS staining, but the stain is of limited utility for leukemia diagnosis, as it is not specific for lymphoblasts.

462. D

AML M7, Acute megakaryoblasticleukemia is the most common AML in DS. However, overall most common leukemias are lymphoid in origin.

463. B

History is suggestive of T-ALL (mediastinal mass is a common presentation, especially in adoloscents), IPT of option B is most appropriate

Option A- undifferentiated leukemia/ lymphoma

Option C- AML

Option D- B cell lymphoma/leukemia

464. C

History is suggestive of DIC (clinical features, low platelets, raised PT and APTT and low fibrinogen) and this is seen most commonly with AML M3 or APML or AML with t(15;17)

465. C

L and H variant, or LP type of cell is seen in NLPHL type.

466. A

HODGKIN LYMPHOMA- classical vs nodular lymphocyte predominant

Marker	Classical RS cells	NLPHL – L and H cells
CD 20	Negative	Positive
CD 30	Positive	Negative
CD 15	Positive	Negative
CD 45	Negative	Positive
CD 10	Negative	Positive
BCL6	Negative	Positive
Pax 5	Weak positive	Strong positive
Oct2/BOB1	Negative	Positive

467. A

Least association of EBV is seen with NLPHL

468. D

To differentiate between benign and malignant lymphocytosis

469. B

Postulated Cells of Origin of Lymphomas Reference- WHO 2017

Lymphoplasma	Postulated normal counterpart (cell of Origin)
Mantle cell lymphoma	B cell of inner mantle zone (Naive B cell) (CD 5 positive)
CLL/SLL	An antigen experienced CD 5 positive cell (either a transformed Naive B cell or memory B cell)
Follicular lymphoma	Germinal centre B cell
Burkitt lymphoma	Germinal centre B cell
DLBCL	Germinal centre B cell or Post germinal centre activated B cell (GCB vs ABC)
Marginal zone lymphoma	Post germinal centre marginal zone (memory B cell)
Lymphoplasmacyt-ic lymphoma	Post germinal centre cell differentiating into plasma cell
Plasma cell myeloma	Plasma cells in which Ig genes have undergone class switching and somatic hypermutation
B-ALL	Hematopoetic stem cell or B cell progenitor
T-ALL	T cell progenitor

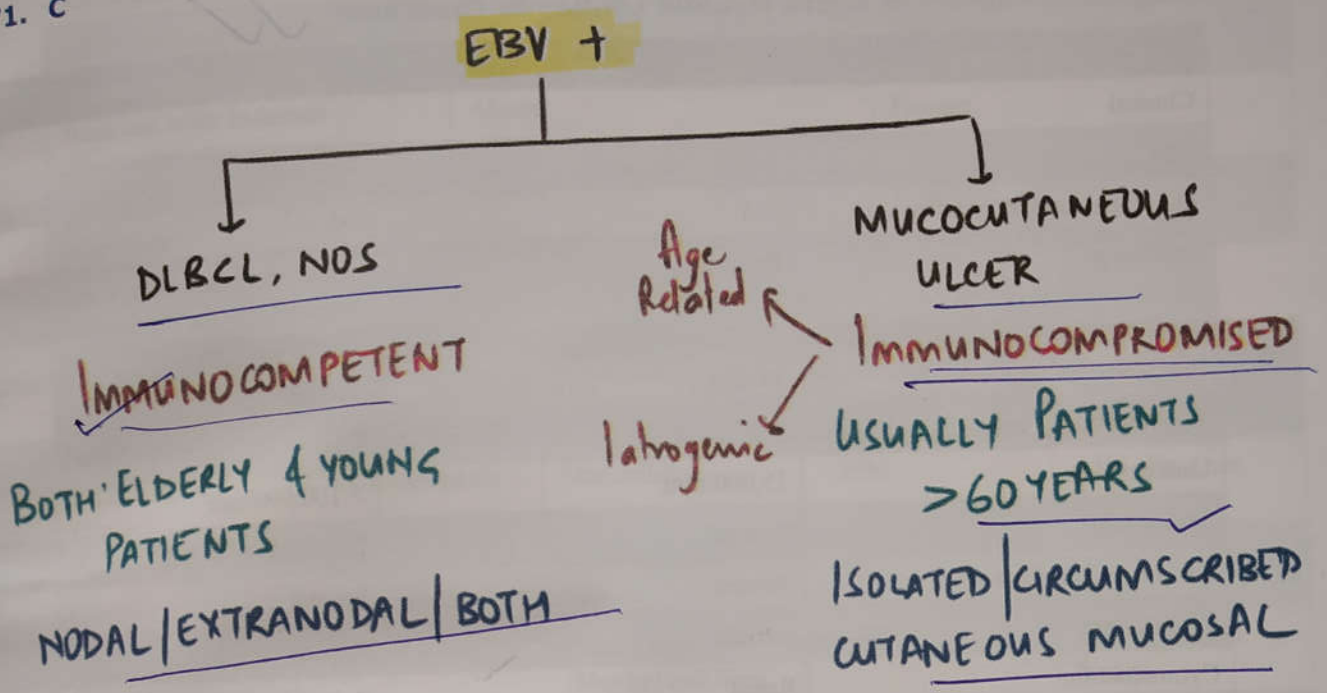
470. A

Option B- Marginal zone lymphoma

Option C- Lymphoplasmacyticlymphoma

Option D- Hairy cell leukemia

471. C



472. A, B, D

B cell transcription factor so absent in option C- T cell condition and in option E- myeloid condition

473. A

Tumor cells stain positive for acid phosphatase which does not go away on washing with tartarate.

474. C

Due to t(11;14)

475. B

Others are lymphomas with retained B cell signature

476. D

To differentiate between benign and malignant polycythemia

477. B

LEBR plus dacrocytes= fibrosis in marrow

478. D

Also in seminoma, GIST and AML
CD 117 positive

479. B

Suggestive for CML

480. B

Auer rods (fused granules) are characteristic of myeloblasts

481. A

Regardless of blast count, AML is diagnosed if one of the following cytogenetic abnormalities are present- t(8;21), t(15;17) and t(16;16) or inv(16)

482. D

CBF alpha or RUNX1T1- option D
CBF beta- options A and B

483. C

Procoagulant molecules made by the tumor cells

484. D

T cell marker
Options A and C- Monocytic differentiation
Option B- stem cell marker

485. C

Prognostic Factors in Acute Myeloid Leukemia Favorable

Factors	Favorable	Unfavorable
Clinical		
Age	< 45y	<2y, >60y
ECOG performance status	0–1	>1
Leukemia	Be novo	Antecedent hematologic disorder; myelodysplasia, myeloproliferative disorder
Infection	Absent	Present
Prior chemotherapy	No	Yes
Leukocytosis	25,000/mm^3	> 100,000/mm^2
Serum LDH	Normal	Elevated
Extramedullar disease	Absent	Present
CNS disease	Absent	Present
Cytoreduction	Rapid	Delayed
Morphology		
Auer rods	Absent	Present
Eosinophils	Absent	Present
Megaloblastic erythroids	Absent	Present
Dysplastic megakaryocytes	Absent	Present
FAB type	M2, M3, M4	M0, M6, M7
Surface/enzyme markers		
Myeloid	CD34-, CD14-, CD13-,	CD34-,
HLA-DR	Negative	Positive
TdT	Absent	Present
Lymphoid	CO2+	CD7+, CD56+ Biphenotypic (2 or more lymphoid markers)
MDR-1	Absent	Present
Cytogenetics	t(16;17), t(8;21), -7, del(7q), -5, del(5q), 3q21 and 3q26 abnormalities inv (16)	complex karyotype
Molecular markers		
Fms-related tyrosine kinase-3 mutation	Absent	Present
Ecotropic viral integration site 1 expression	Absent	Present
Mixed-lineage leukemia partial tandem duplication	Absent	Present

Nucleophosmin mutation	Present	Absent
CCAAT/enhancer-binding protein-a mutation	Present	Absent
Brain and acute leukemia cytoplasmic gene expression	Absent	Present
Vascular endothelial growth factor expression	Absent	Present

486. A

	Blood Cytopenia (c)	Bone Marrow Dysplasia (> 10%)	Ringed Sideroblasts (Marrow)	Blasts in marrow
MDS – SLD (Single)	UN1/Bicytopenia	One cell line	< 15%	< 5% (No Aner Rods)
MDS – MLD (Multi)	UNI To Pancytopenia	More than one cell line	< 15%	< 5% (No Aner Rods)
MDS – RS – SLD (Single)	UNI Cytopenia	One or two (Maybe) cell lines	>15%	< 5% (No Aner Rods)
MDS – RS – SLD (Multi)	UNI To Pancytopenia	More than one cell line	> 15%	< 5% (No Aner Rods)
MDS – EB1 (Excess Blasts)	UNI To Pancytopenia	One or three cell lines	None or Any	5 -9% (No Aner Rods)
MDS – EB2	UNI To Pancytopenia	One or three cell lines	None or Any	10 -19% or Aner Rods

487. B

CML-AP is defined by the presence of £ 1 of the following haematological/cytogenetic criteria or provisional criteria concerning response to tyrosine kinase inhibitor (TKI) therapy

Haematologica! /cytogenetic criteria[8]

- Persistent or increasing high white blood cell count (> 10 × 10^9/L), unresponsive to therapy
- Persistent or increasing splenomegaly, unresponsive to therapy
- Persistent thrombocytosis (>1000 × 10^9/L), unresponsive to therapy
- Persistent thrombocytopenia (< 100 × 10^9/L), unrelated to therapy
- £20% basophils in the peripheral blood
- 10—19% blasts in the peripheral blood and/or bone marrow[b,c]
- Additional clonal chromosomal abnormalities in Philadelphia (Ph) chromosome-positive (Ph+) ceils at diagnosis, including so-called major route abnormalities (a second Ph chromosome, trisomy 8, isochromosome 17q, trisomy 19), complex karyotype, and abnormalities of 3q26,2
- Any new clonal chromosomal abnormality in Ph+ ceils that occurs during therapy

Provisional response-to-TKI criteria

- Haematological resistance (or failure to achieve a complete haematologica! responsed) to the first TKI
- Any haematological, cytogenetic, or molecular indications of resistance to two sequential TKIs
- Occurrence of two or more mutations in the BCR-ABL1 fusion gene during TKI therapy

488. B

Option A- Burkitt lymphoma

Option C- APML

Option D- Mantle cell lymphoma and Multiple myeloma

489. B

Option A- Follicular lymphoma

Option C- T cell lymphomas

Option D- HL, PTCL and ALCL

490. A

Option C- Dutcher bodies

491. A

Ann Arbor staging system is used

492. D

Clinical Risk Factor	Point per Risk Factor
Age >60	1
serum LDH>normal	1
performance status 2-4	1
stage 3 or 4	1
extra nodal involvement >1	1

Risk for CNS involvement based on total points: 0 - 1 points is low risk, 2 points is low to intermediate risk, 3 points is intermediate to high risk, and 4 or 5 points is high risk.

493. A

Diagnosis is CLL

Option A- pathognomonic for CLL

494. C

B cell lymphoma

Option A- positive in MCL and CLL

Option B- HL

Option D- Mastocytosis

495. A

Option B- most common site in immunocompetent for extranodal NHL

496. B

Usually EBV induced (not CMV)

497. B

Option A- gum hypertrophy

Option D- most common in Down's syndrome and causes myelofibrosis

498. D

Myeloid lineage

MPO (by flow cytometry, immunohistochemistry, or cytochemistry) or

Monocytic differentiation (>2 of the following: non-specific esterase, CD11c, CD14, CD64, lysozyme)

T-cell lineage

Cytoplasmic CD3 (by flow cytometry with antibodies to CD3 epsilon chain. Immunohistochemistry using polyclonal anti-CD3 antibody may detect CD3 zeta chain, which is not T-cell-specific)

or

Surface CD3 (rare in mixed-phenotype acute leukaemias)

B-cell lineage (multiple antigens required)

Strong CD19

with > 1 of the following strongly expressed: CD79a, cytoplasmic CD22, CD10

or

WeakCD19

with >2 of the following strongly expressed: CD79a, cytoplasmic CD22, CD10

499. C

Due to unexplained monocytopenia

500. A

Cutaneous T cell lymphoma

501. B (single best option)

An M protein is found in the serum or urine in about 97% of cases: IgG in 50% of these cases; IgA in 20%; light chain in 20%; and IgD, IgE, IgM, or biclonal in < 10%. About 3% of cases are non-secretory. The serum M protein is usually > 30 g/L of IgG and > 20 g/L of IgA. In 90% of patients, there is a decrease in polyclonal Ig(< 50% of normal). Other laboratory findings include hypercalcaemia (found in up to

10% of cases), elevated creatinine (in 20- 30%), hyperuricaemia (in > 50%), and hypoalbuminaemia (in - 15%).

502. D

Intracytoplasmic Ig Inclusions

503. D

Lymphoplasmacytic lymphoma is a B-cell neoplasm of older adults that usually presents in the sixth or seventh decade of life. Although bearing a superficial resemblance to CLL/SLL, it differs in that a substantial fraction of the tumor cells undergo terminal differentiation to plasma cells. Most commonly, the plasma cell component secretes monoclonal IgM, often in amounts sufficient to cause a hyperviscosity syndrome known as Waldenström macroglobulinemia. Unlike multiple myeloma, complications stemming from the secretion of free light chains (e.g., renal failure and amyloidosis) are relatively rare and bone destruction does not occur. Recent deep sequencing studies have shown that virtually all cases of lymphoplasmacytic lymphoma are associated with acquired mutations in MYD88. The MYD88 gene encodes an adaptor protein that participates in signaling events that activate NF-κB and also augment signals downstream of the B-cell receptor (Ig) complex, both of which may promote the growth and survival of the tumor cells

504. C

Extranodal NK/T celllymphoma

505. C

Reference WHO 2017

Myeloid lineage

MPO (by flow cytometry, immunohistochemistry, or cytochemistry) or

Monocytic differentiation (>2 of the following: non-specific esterase, CD11c, CD14, CD64, lysozyme)

T-cell lineage

Cytoplasmic CD3 (by flow cytometry with antibodies to CD3 epsilon chain. Immunohistochemistry using polyclonal anti-CD3 antibody may detect CD3 zeta chain, which is not T-cell-specific)

or

Surface CD3 (rare in mixed-phenotype acute leukaemias)

B-cell lineage (multiple antigens required)

Strong CD19

with > 1 of the following strongly expressed: CD79a, cytoplasmic CD22, CD10

or

WeakCD19

with >2 of the following strongly expressed: CD79a, cytoplasmic CD22, CD10

506. B

The marked areas show lymphoid follicles which are rich in B cells, highlighted by CD 20. CD 3 is a T cell marker. CD 56 is a marker for NK cells. CD 68 is a marker for histiocytic cells.

507. D

The clinical features and electron microscopy picture are consistent with Hand Schuller Christian triad- which is a multifocal unisystem occurrence of Langerhan's cell histiocytosis. The markers for LCH are all mentioned (CD 207 is Langerin)

508. B

509. A

510. A, B, E

511. A

512. A, B, C, D, E

513. A

514. D

515. B

516. D

517. C

518. C

519. D

520. B

521. D
522. C
523. C
524. A
525. C
526. C
527. A
528. B
529. C
530. C
531. D
532. B
533. A
534. C
535. B
536. B
537. C
538. B
539. D
540. A
541. A
542. D
543. D
544. D
545. B
546. B
547. B
548. A
549. C
550. C
551. C
552. D
553. A
554. A, C
555. D
556. D
557. C
558. A
559. B
560. A
561. A
562. D
563. D
564. B
565. B
566. C
567. B
568. C
569. B
570. A
571. B
572. A
573. A
574. B
575. C

Platelet Disorders – Questions

576. Bleeding time increased in which of the following conditions?

a. Von Willebrand disease
b. Hemophilia A
c. DIC
d. Both a & c

577. Which of the following is true regarding Bernard-Soulier syndrome?

a. It is due to defect in platelet adhesion
b. It is due to defect in platelet aggregation
c. It is due to defect in platelet receptor GpIb-IX
d. Both a and c

578. Which of the following is/are released from dense granules of platelets? (PGI)

a. Serotonin
b. Histamine
c. PDGF
d. ATP
e. Lysosome

579. In PT test, the addition of Ca^{2+} & tissue thromboplastin activates which pathway?

a. Extrinsic
b. Intrinsic
c. Fibrinolytic
d. Common

580. Both PT and aPTT will be increased in deficiency of? (PGI)

a. Factor 2
b. Factor 5
c. Factor 8
d. Factor 10
e. Factor 12

581. Which of the following is a vitamin K dependent clotting factor? (PGI)

a. Factor XII
b. Factor II
c. Factor VII
d. Factor X
e. Factor IX

582. Von Willebrand disease, all are true except-

a. Factor VIII C deficiency
b. BT prolonged
c. Normal ristocetin test
d. Defective aggregation

583. In Glanzmann thromboasthenia there is a defect in-

a. Platelet aggregation
b. Platelet adhesion
c. Decreased ADP release
d. Disordered platelet secretion

584. A newborn baby presented with profuse bleeding from the umbilical stump after birth. Rest of the Examination and PT, APTT are within normal limits. Most of probable diagnosis is-

a. Factor X deficiency
b. Glanzmann thrombasthenia
c. Von Willebrand disease
d. Bernard-Soulier disease

585. What is not associated with DIC-

a. Thrombocytopenia
b. Increased PT
c. Hyperfibrinogenemia
d. Increased FDP

586. Earliest event of vascular trauma is?

a. Vasoconstriction
b. Platelet adhesion
c. Platelet aggregation
d. Vasodilatation

587. PDGF is present in which granules of platelets?

a. Alpha
b. Beta
c. Delta
d. None

588. Which of the following is true regarding Von willebrand disease?

a. Type 1&3 are associated with quantitative defects in vWF
b. Normal platelet count
c. Desmopressin stimulates release of VWF
d. All of the above

576. d	577. d	578. a,b	579. a	580. a,b,d	581. b,c,d,e	582. c	583. a
584. b	585. c	586. a	587. a	588. d			

589. Which of the following is the most common manifestation of haemophilia?
a. Hemoptysis
b. Hemarthrosis
c. Hematemesis
d. Mucosal bleeding

590. Which coagulation factor is not in circulating form in blood?
a. F XI
b. F X
c. F III
d. F XIII

591. Which of the following is not associated with prolonged prothrombin time? (PGI)
a. Haemophilia A
b. Von-Willebrand disease
c. Factor VII deficiency
d. Disseminated intravascular coagulation (DIC)

592. Feature(s) of XIII factor deficiency is/are: (PGI)
a. Delayed wound closure
b. Clot solubility tests are abnormal
c. ↑aPTT
d. ↑PT
e. ↑BT

593. Endotoxin first damages?
a. Endothelium
b. RBC
c. Platelets
d. WBC

594. Which of the following measures the intrinsic pathway?
a. CT
b. aPTT
c. TT
d. Prothrombin time

595. An 11 years old boy has elevated prothrombin and activated partial prothrombin time. What is the most likely defect?
a. Defect in extrinsic pathway
b. Defect in intrinsic pathway
c. Defect in common pathway
d. Defect in platelet function

596. Which of the following is true regarding Factor V Leiden mutation?
a. Increased risk of deep vein thrombosis
b. Factor V becomes resistant to cleavage by protein C
c. Glutamine to Arginine substitution at 506
d. All of the above

597. Where can Von Willebrand factor (VWF) be found in the body?
a. Delta granules of platelets
b. Weibel-Palade bodies
c. Kupffer cells
d. Merkel cells

598. What is the most common inherited bleeding disorder?
a. Hemophilia A
b. Hemophilia B
c. Von Willebrand disease
d. Factor XI deficiency

599. Which factors are part of the intrinsic system of the coagulation cascade?
a. II, VII, IX, X
b. VII, TF
c. X, V, II, I
d. VIII, IX, XI, XII
e. II, V, VIII, X

600. Which coagulopathy is associated with amyloidosis?
a. Factor V inhibitor
b. Factor X deficiency
c. Factor VIII inhibitor
d. Factor V deficiency
e. Plasminogen activator inhibitor-1

601. Which factor has the shortest plasma half-life?
a. Factor IX
b. Factor XI
c. Factor VII
d. Factor I
e. Factor X

602. Which of the following conditions has the highest incidence of disseminated intravascular coagulation (DIC)?
a. Solid tumors
b. Aortic aneurysm
c. Advanced lung cancer
d. Sepsis/severe infection

589. b	590. c	591. a,b	592. a,b	593. a	594. b	595. c	596. d	597. b
598. c	599. d	600. b	601. c	602. d				

603. Activated protein C resistance due to Factor V Leiden is caused by which of the following?

a. A point mutation in Factor V
b. An autoantibody to activated protein C
c. A deletion in protein C
d. A point mutation in protein C
e. Abnormal thrombin binding to Factor V

604. Major source of von Willebrand factor-

a. Erythrocytes
b. Neutrophils
c. Endothelial cells
d. Monocytes

605. Hemophilia A has following except:

a. VIII factor ↓ b. PTT ↑
c. PT ↓ d. Normal BT

606. The presence of small sized platelets on the peripheral smear is characteristic of-

a. Idiopathic thrombocytopenic purpura
b. Bernard-Soulier syndrome
c. Disseminated intravascular coagulation
d. Wiskott-Aldrich syndrome

607. Which is not true regarding Bernard – Soulier syndrome?

a. Ristocetin aggregation is normal
b. Aggregation with collagen and ADP is normal
c. Large platelets
d. Thrombocytopenia

608. Which of the following statements about platelet function defects is true?

a. Normal platelet count with prolonged bleeding time
b. Thrombocytopenia with prolonged bleeding time
c. Thrombocytosis with prolonged bleeding time
d. Normal platelet count with normal bleeding time

609. In DIC, following are seen except-

a. Fibrinogen decreased
b. Thrombocytopenia
c. Normal aPTT
d. PT elevation

610. Cryoprecipitate is useful in-

a. Hemophilia B
b. Thrombasthenia
c. Afibrinogenemia
d. Warfarin reversal

611. All of the following are surface bound zymogens except

a. Hageman factor
b. Christmas factor
c. Fletcher factor
d. Factor XI

612. All of the following are features of primary hemostasis except

a. Delayed onset post trauma
b. Sites affected are skin and mucous membranes
c. Thrombocytopenia is a clinical example
d. Deep tissues are usually not involved

613. PT and aPTT are normal in all of the following except

a. Von Willebrand disease
b. Factor XIII deficiency
c. Lupus anticoagulant
d. Vascular disorder of hemostasis

614. Most common cause of bleeding caused by platelet dysfunction in a myeloproliferative neoplasm is

a. Polycythemia vera
b. Idiopathic myelofibrosis
c. Chronic myeloid leukemia
d. Essential thrombocytosis

615. A male patient with history of bleeding was investigated and was found to have increased APTT but normal PT. His maternal uncle had a similar presentation. The next step of investigation should be

a. Factor VIII assay
b. Dilute Russell viper venom test
c. Mixing studies
d. Factor IX assay

603. a	604. c	605. c	606. c	607. a	608. a	609. c	610. c	611. b	612. a
613. c	614. a	615. c							

Platelet Disorders – Explanations

576. D

Option C- coagulation factor defect

Bleeding time is a laboratory test to assess platelet function and the body's ability to form a clot. The test involves making a puncture wound in a superficial area of the skin and monitoring the time needed for bleeding to stop (ie, bleeding site turns "glassy").

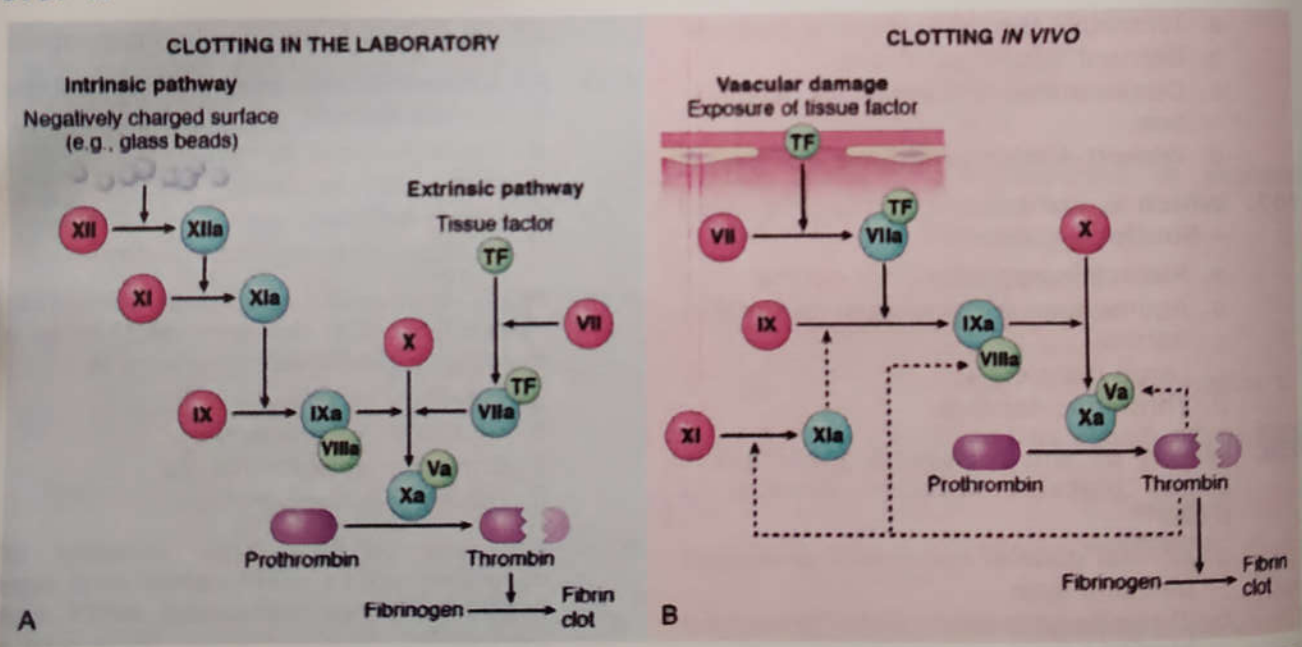
577. D

Bernard-Soulier syndrome illustrates the consequences of defective adhesion of platelets to subendothelial matrix. Bernard-Soulier syndrome is caused by an inherited deficiency of the platelet membrane glyco protein complex Ib-IX. This glycoprotein is a receptor for vWF and is essential for normal platelet adhesion to the subendothelial extracellular matrix. Affected patients have a variable, often severe, bleeding tendency

578. A, B

α-Granules have the adhesion molecule Pselectin on their membranes (Chapter 3) and contain proteins involved in coagulation, such as fibrinogen, coagulation factor V, and vWF, as well as protein factors that may be involved in wound healing, such as fibronectin, platelet factor 4 (a heparinbinding chemokine), plateletderived growth factor (PDGF), and transforming growth factorβ. Dense (or δ) granules contain adenosine diphosphate (ADP) and adenosine triphosphate, ionized calcium, serotonin, and epinephrine.

579. A



Coagulation factor	PT	APTT
Fibrinogen (Factor I)	Increased	Increased
Prothrombin (Factor II	Increased	Increased
Labile factor/ Proaccelerin (Factor V)	Increased	Increased

Stable factor/Proconvertin (Factor VII)	Increased	Normal
Anti hemophilic factor (Factor VIII)	Normal	Increased
Christmas factor (Factor IX)	Normal	Increased
Stuart Power factor (Factor X)	Increased	Increased
Plasma thromboplastin antecedent (Factor XI)	Normal	Increased
Hageman factor (Factor XII)	Normal	Increased
Fibrin stabilising factor (Factor XIII)	Normal	Normal

580. A, B, D

581. B, C, D, E

Surface bound zymogens
Factor XII (Hageman factor)
Prekallikrein (Fletcher factor)
Factor XI
Vitamin K dependent zymogens (phospholipid bound)
Factor X
Factor IX (Christmas factor)
Factor VII
Factor II (Prothrombin)
Protein C (acts as an inhibitor when activated)
Cofactors or substrates
HMWK (Fitzgerald factor or Williams factor)
Factor VIII (antihemophilic factor)
Factor V
Fibrinogen
Protein S (Vitamin K dependent cofactor for activated Protein C)

582. C

Patients with von Willebrand disease have defects in platelet function despite a normal platelet count. The plasma level of active vWF, measured as the ristocetin cofactor activity, is reduced. Because vWF stabilizes factor VIII, a deficiency of vWF gives rise to a secondary decrease in factor VIII levels. This may be reflected by a prolongation of the PTT in von Willebrand disease types 1 and 3. However, except in rare type 3 patients, adverse complications typical of severe factor VIII deficiency, such as bleeding into the joints, are not seen. Even within families in which a single defective vWF allele is segregating, wide variability in clinical expression is common. This is due in part to modifying genes that influence circulating levels of vWF, which show a wide range in normal populations. Persons with von Willebrand disease facing hemostatic challenges (dental work, surgery) can be treated with desmopressin, which stimulates vWF release, or with infusions of plasma concentrates containing factor VIII and vWF.

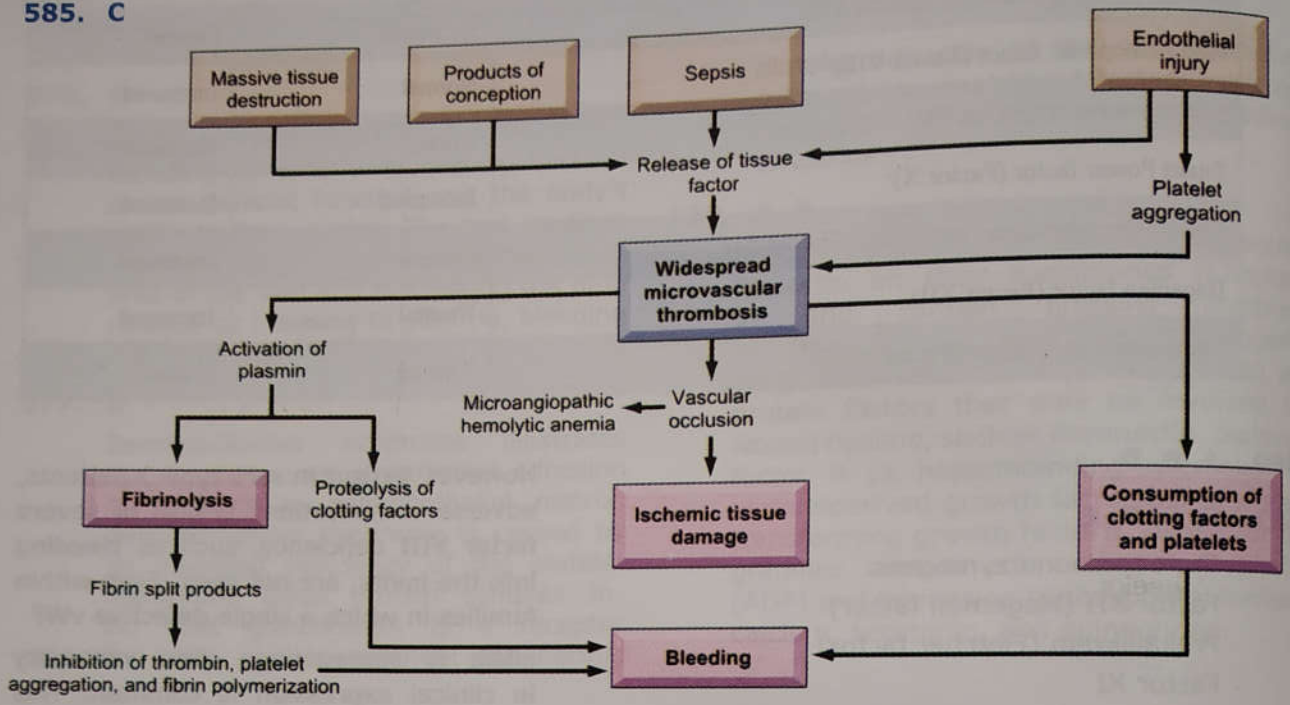
583. A

Bleeding due to defective platelet aggregation is exemplified by Glanzmann thrombasthenia, which is also transmitted as an autosomal recessive trait. Thrombasthenic platelets fail to aggregate in response to adenosine diphosphate (ADP), collagen, epinephrine, or thrombin because of deficiency or dysfunction of glycoprotein IIbIIIa, an integrin that participates in "bridge formation" between platelets by binding fibrinogen. The associated bleeding tendency is often severe

584. B

Option A- increased PT and APTT

585. C



586. A
587. A
588. D
589. B
590. C
591. A, B
592. A, B
593. A
594. B
595. C
596. D
597. B
598. C
599. D
600. B

601. C
602. D
603. A
604. C
605. C
606. D
607. A
608. A
609. C
610. C
611. B
612. A
613. C
614. A
615. C

Transfusion Medicine – Questions

616. Blood group AB is denoted by which colour label

a. Pink
b. Red
c. White
d. Yellow

617. All are true about FFP except

a. Frozen within 8 hours of collection of whole blood
b. 240 ml plasma in one unit
c. Can be stored for 5 years
d. Minimal loss of labile coagulation factors

618. Interval between 2 blood donations should be at least

a. 48 hours
b. 6 weeks
c. 12 weeks
d. 20 weeks

619. Platelets can be stored at 22-25 deg C with agitation for how many days?

a. 5 days
b. 7 days
c. 21 days
d. 35 days

620. Patient comes to ER with severe bleeding. Blood group is not known. Which group FFP will you give to the patient

a. O +ve
b. O –ve
c. Ab +ve
d. A +ve

621. Patient with severe bleeding comes to emergency dept. As per your demand you get 4 platelet and 2 PRC units from blood bank. What should be the next step

a. Transfuse PC and store PRC at room temp
b. Transfuse PC and store PRC between 2-4 degrees
c. Transfuse PRC and store PC at room temp
d. Transfuse PRC and store PC in 2-4 degrees

622. All are true about cryoprecipitate except

a. Thawed at -4 deg C
b. 120 units of factor VIII
c. 150 mg of Fibrinogen
d. Can be used for Hemophilia A

623. TRALI is seen within __ hours of transfusion

a. 4
b. 6
c. 8
d. 10

624. Most common complication of blood transfusion is

a. Hypothermia
b. Hypokalemia
c. Hyperkalemia
d. Hypocalcemia

625. Function of glucose in stored blood is

a. Isotonicity
b. Nutrition
c. Preventing hemolysis
d. Maintain ATP

626. Any transfusion should be completed within __ hours of initiation

a. 4 hours
b. 6 hours
c. 8 hours
d. 10 hours

627. All of the following components should be mandatorily tested for compatibility except

a. RBCs
b. Cryoprecipitate
c. FFP
d. Platelets

616. c	617. c	618. c	619. a	620. c	621. c	622.	623. b	624. a	625. c
626. a	627. b								

628. In which of the following scenarios is blood radiated

a. BMT patient
b. Blood relative is a donor
c. Following intra uterine transfusion
d. All of the above

629. Most common reaction associated with blood transfusion is

a. Febrile non hemolytic transfusion reaction
b. TRALI
c. GVHD
d. Delayed hemolytic reactions

630. All are true about post transfusion purpura except

a. >5 days after platelet transfusion
b. More in women
c. Platelet specific antibodies against HPA-1a
d. Additional platelet transfusion is not helpful

631. All are true about transfusion in a patient with IgA deficiency except

a. More risk of anaphylaxis with plasma transfusion
b. IgA rich plasma given
c. <1% population affected
d. Washed cellular components given

632. Genes for A and B antigens are on

a. 9p
b. 9q
c. 19p
d. 19q

633. Interval between 2 plasma pharesis should be

a. 48 hours
b. 6 weeks
c. 12 weeks
d. 20 weeks

634. Shelf life of RBCs stored in CPDA is

a. 21 days
b. 28 days
c. 35 days
d. 42 days

635. Platelet transfusion should be completed within

a. 60 minutes
b. 4 hours
c. 6 hours
d. 8 hours

628. d	629. a	630. a	631. b	632. a	633. a	634. c	635. a

616. C

The following colour code is used to differentiate the ABO group label

- Blood group O - Blue
- Blood group B - Pink
- Blood group A - Yellow
- Blood group AB – White

617. C

- Fresh frozen plasma (FFP) is prepared by separating citrated plasma from whole blood and freezing it within 8 hours of collection or by freezing citrated apheresis plasma within 6 hours of collection.
- Each unit of FFP prepared from whole blood contains approximately 200 ml of plasma.
- Apheresis plasma may be packaged into 200- or 400-ml bags.
- FFP may be stored at -18°C or below for up to 1 year.
- Under these conditions, there is minimal loss of activity of the labile coagulation factors V and VIII.
- One milliliter of FFP contains approximately one unit of coagulation factor activity. After thawing,
- FFP may be stored in the refrigerator for up to 24 hours before use

618. C

- The interval between two blood donations should be at least 12 weeks.
- At least 48 hours must elapse after plasma pheresis or Cytapheresis before whole blood is collected from a donor
- Apheresis should be done only after 90 days of whole blood collection or in an event when red cells are not returned at the end of pheresis

619. A

- Platelets should be stored at 22-24° C (controlled temperature) with continuous gentle agitation in platelet incubator and agitator.
- Can be stored for upto 5 days
- ABO compatibility needed whenever possible

620. C. AB +ve

Fresh Frozen Plasma

- FFP is plasma prepared from whole blood, either from the primary centrifugation of whole blood into red cells and plasma or from a secondary centrifugation of platelet rich plasma.
- The plasma is rapidly frozen to −25°C or colder within 8 hours of collection and contains normal plasma levels of stable clotting factors, albumin, immunoglobulin and
- Factor VIII at a level of at least 70% of normal fresh plasma.
- Should be ABO compatible.

Universal Plasma Donor

- Plasma components should be compatible with the ABO group of the recipient to avoid potential haemolysis caused by donor anti-A or anti-B.
- Group AB individuals have neither anti-A nor anti-B antibodies in their plasma. Group AB plasma can therefore be given to patients of any ABO blood group and is often referred to as the universal plasma donor

Choice of plasma transfusion

Component ABO Group				
Recipient ABO Group	1st Choice	2nd Choice	3rd Choice	4th Choice
AB	AB	(A)	(Bj	(0)
A	A	AB	(B)	(0)
B	8	AB	(A)	(0)
0	0	A	B	AB

621. C

This is a straight forward practical question. In a severely bleeding patient first we have to transfuse PRBC.

Now we will look at the platelets.

- If you have a place to store platelets, platelets can be transfused without agitation in room temperature upto 24 hours.
- Seeing the options, the it seems there is a facility to store platelets.
- So PC will be the second to be transfused. In a setting where you don't have storing option (like hospital ward), platelets should be transfused first for concern of bacterial contamination (since two options in the above questions are about storing platelets we presume there is a facility to store platelets)

622. A. Cryoprecipitated AHF, OR Cryo-precipitate

- An extract of FFP that is enriched in high-molecular-weight plasma proteins.
- It is prepared by thawing one unit of FFP at 1°C to 6°C
- Each unit of this cryoprecipitate contains approximately 80 to 120 units of Factor VIII and at least 150 mg of fibrinogen.
- It also contains factor XIII, fibronectin, and the high-molecular-weight multimers of vWF

623. B

TRALI

- Most common cause of death following blood transfusion
- Occurs within 6 hours of transfusion
- Most associated with plasma products
- Anti HLA class 1 antibodies and anti granulocyte antibodies

624. A

Complications of Massive Blood Transfusion-

- Hypothermia (most common complication) (cold fluid infusion and no thermal regulation)
- Hypokalemia (re entry into transfused RBCs, and stress hormone release) > Hyperkalemia (stored blood has more potassium and there is potassium retention due to loss of hydrogen ions in response to acidosis)
- Metabolic alkalosis (citrate metabolism to bicarbonate) > acidosis
- Hypocalcemia (rapid transfusion can cause citrate overload)
- Hypomagnesemia (magnesium poor fluid)
- Dilutional coagulopathy (most common cause of death following massive blood transfusion)

625. C

Red Cells

Additives

- Sodium chloride: Isotonicity
- Mannitol: reduces cell lysis
- Dextrose: nutrition
- Glucose: osmotic agent to reduce hemolysis
- Phosphate: buffer solution
- Adenine: for ATP maintenance, increases shelf life
- Citrate: anti coagulant

- One unit of red cells is often given in 1 to 4 hours, depending on the amount to be transfused and on the patient's cardiovascular status.

Infusion of a unit for longer than 4 hours is not recommended, as there is a risk of bacterial proliferation because the opened unit is at room temperature.

Storage times

- CPD : 21 days
- CPDA : 35 days
- SAGM : 42 days

626. A

- In general, it is desirable to complete a red cell transfusion within 2 hours (not exceeding 4 hours) and a platelet or plasma transfusion within 30–60 minutes.
- Any transfusion should be completed within 4 hours of initiation.

627. B

- Recipient should receive ABO type specific compatible whole blood or red blood cell components.
- In the absence of ABO type specific blood, group O packed red cells should be transfused.
- Platelet concentrate should be ABO and Rh(D) type specific with the recipient blood as far as possible
- Leucocyte concentrate should be ABO and Rh(D) type specific or compatible with the recipient blood
- ABO compatibility not mandatory for cryoprecipitate but mandatory for fresh frozen plasma

628. D

Irradiation

Cellular components should be irradiated in order to reduce the risk of post transfusion GVHD when a patient is identified as being at risk for GVHD

- For all immunosuppressed patients including bone marrow transplant (BMT) patients
- When blood from a blood relative is used.
- In case of exchange transfusion following intra uterine transfusion.

The minimum dose delivered to the blood bag should be 25 Gy + 2

629. A

The most frequent reaction associated with the transfusion of cellular blood components is a febrile nonhemolytic transfusion reaction (FNHTR). These reactions are characterized by chills and rigors and a more than 1 deg C rise in temperature. FNHTR is diagnosed when other causes of fever in the transfused patient are ruled out. Antibodies directed against donor leukocyte and HLA antigens may mediate these reactions; thus multiply transfused patients and multiparous women are felt to be at increased risk

630. A

This reaction presents as thrombocytopenia 7 – 10 days after platelet transfusion and occurs predominantly in women. Platelet-specific antibodies are found in the recipient's serum, and the most frequently recognized antigen is HPA-la found on the platelet glycoprotein IIIa receptor. The delayed thrombocytopenia is due to the production of antibodies that react to both donor and recipient platelets. Additional platelet transfusions can worsen the thrombocytopenia and should be avoided. Treatment with intravenous immunoglobulin may neutralize the effector antibodies, or plasmapheresis can be used to remove the antibodies.

631. B

Patients who are IgA-deficient, < 1 % of the population, may be sensitized to this Ig class and are at risk for anaphylactic reactions associated with plasma transfusion. Individuals with severe IgA deficiency should therefore receive only IgA-deficient plasma and washed cellular blood components. Patients who have anaphylactic or repeated allergic reactions to blood components should be tested for IgA deficiency.

632. A

The first blood group antigen system-recognized in 1900 was ABO, the most important in transfusion medicine. The major blood groups of this system are A, B, AB, and O. 0 type RBCs lack A or B antigens. These antigens are carbohydrates attached to a precursor backbone, may be found on the cellular membrane either as glycosphingolipids or glycoproteins, and are secreted into plasma and body fluids as glycoproteins. H substance is the immediate precursor on which the A and B antigens are added. This H substance is formed by the addition of fucose to the glycolipid or glycoprotein backbone. The subsequent addition of N-acetylgalactosamine creates the A antigen, whereas the addition of galactose produces the B antigen. The genes that determine the A and B phenotypes are found on chromosome 9p and are expressed in a Mendelian codominant manner. The gene products are glycosyl transferases, which confer the enzymatic capability of attaching the specific antigenic carbohydrate Individuals who lack the "A" and "B" transferases are phenotypically type "0," whereas those who inherit both transferases are type "AB." Rare individuals lack the H gene, which codes for fucose transferase, and cannot form H substance. These individuals are homozygous for the silent h allele (hh) and have Bombay phenotype (Oh)'

633. A

634. C

635. A

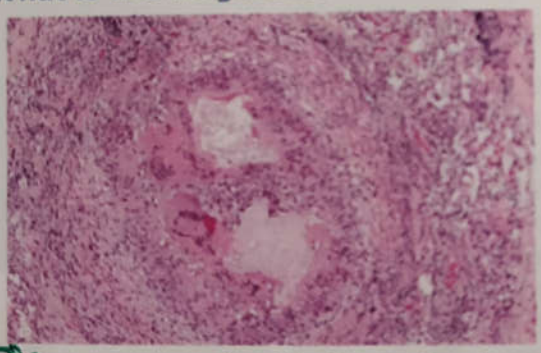
636. Loss of function mutations in which of the following leads to Hirschsprung disease

a. MET
b. RAS
c. Rb
d. RET

637. A 56-years-old chronic smoker, mass in bronchus resected. What is the possible marker to confirm diagnosis?

a. Cytokeratin
b. Vimentin
c. Epithelial membrane antigen
d. Leukocyte common antigen

638. Patient with h/o long standing depressive illness come to emergency with acute breathlessness. The X-ray shows diffuse infiltrates with predominance in right middle lobe and right lower lobe. The patient did not survive and the following picture in the lungs was seen on autopsy. What is the diagnosis?



a. Severe necrosis with fungal hyphae
b. Caseous necrosis
c. Vegetable matter- Aspiration pneumonia
d. Severe necrotizing pneumonia

639. A patient underwent lung transplantation. The resected lung from the patient showed followig features. What could be your possible diagnosis?

a. Bronchiectasis
b. Lung abscess
c. Lung carcinoma
d. Miliary tuberculosis

640. Endotoxin shock is initiated by-

a. Endothelial injury
b. Peripheral vasodilation
c. Increased vascular permeability
d. Cytokines action

641. Silicosis biopsy which is true about features and radiological correlation? (PGI)

a. Lower lobe involved
b. Dense collagen and calcifications in the lymph nodes seen
c. Progressive massive fibrosis can be seen as late complication
d. Immune granuloma can be seen
e. Macules may be seen

642. WHO 2015 new inclusion in lung squamous cell carcinoma is/are?

a. Basaloid type
b. Lymphoephtheliod type
c. Papillary type
d. Clear cell variety
e. Small cell variety

636. d	637. a	638. c	639. a	640. d	641. b	642. a

643. Which one of the following markers is/are useful in differentiating between mesothelioma and adenocarcinoma- (PGI)

a. CEA
b. Cytokeratin
c. Calretinin
d. TTF
e. Vimentin

644. Lung Ca metastasize early is?

a. Adeno ca
b. Sq cell Ca
c. Small cell Ca
d. Large cell ca

645. Which of these is used as a marker in mesothelioma? (PGI)

a. Calretinin
b. TTF-1
c. CK-5/6
d. Glypican
e. D240

646. The highest malignant potential is seen in-

a. a.Crohn's disease
b. Ulcerative colitis
c. FAP
d. Peutz Jegher syndrome

647. Macrophages containing PAS positive granules and rod-shaped bacilli in small intestinal mucosa are found in-

a. Agammaglobulinemia
b. Tropical sprue
c. Whipple's disease
d. Coeliac spure

648. Ectopic ACTH production is seen in:

a. Small cell carcinoma of lung
b. Anaplastic carcinoma of lung
c. Squamous cell carcinoma of lung
d. Adenocarcinoma of lung

649. 1-antitrypsin deficiency is associated with-

a. Centriacinar emphysema
b. Panacinar emphysema
c. Paraseptal emphysema
d. Irregular emphysema

650. Ferruginous bodies are seen in-

a. Silicosis
b. Bysinosis
c. Asbestosis
d. Bagassosis

651. Bilateral hilar lymphadenopathy with non caseating granuloma is seen in-

a. TB
b. Lymphoma
c. Sarcoidosis
d. All of the above

652. A 40-year-old alcoholic man is admitted to the hospital in severe respiratory distress. The temperature is 38.7°C (103°F), respirations are 32 per minute, and blood pressure is 130/90 mm Hg. He coughs constantly and expectorates "currant-jelly" sputum. A chest X-ray reveals bilateral diffuse pulmonary consolidation. Physical examination shows bilateralcrackles, dullness to percussion over both pulmonary fields, and use of accessory muscles. The patient subsequently dies from complications of bacterial sepsis. The lung at autopsy is shown below. What is the appropriate diagnosis?

a. Atypical pneumonia
b. Bronchopneumonia
c. Lobar pneumonia
d. Pulmonary abscess

643. a,c,d,e	644. c	645. a,c,e	646. c	647. c	648. a	649. b
650. c	651. c	652. c				

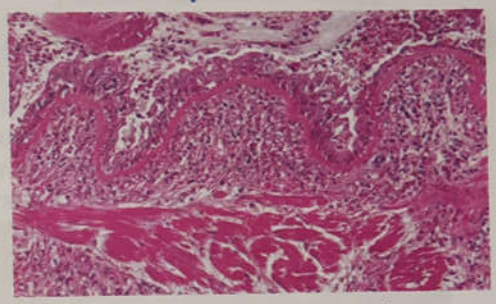
653. A 36-year-old man with AIDS presents with fever, dry cough,and dyspnea. A chest X-ray shows bilateral and diffuse infiltrates.Laboratory studies reveal a CD4+ cell count of less than 50/L. A lung biopsy discloses a chronic interstitial pneumonitis and an intra-alveolar foamy exudate. A silver stain ofa bronchoalveolar lavage is shown in the image. Which of the following organisms is the most likely pathogen responsible for these pulmonary findings?

a. Cryptococcus
b. Histoplasma
c. Pneumocystis
d. Mycobacterium

654. A 35-year-old woman with a long history of dyspnea, chroniccough, sputum production, and wheezing dies of respiratoryfailure following a bout of lobar pneumonia. She was a non-smoker and did not drink alcoholic beverages. The lung atautopsy is shown in the image. Which of the following underlyingconditions was most likely associated with the pathologic changes shown here

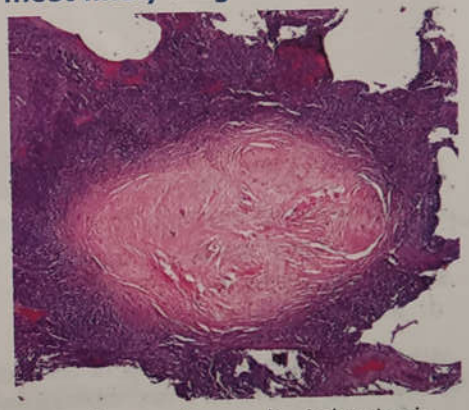
a. Alpha 1 antitrypsin deficiency
b. Cystic fibrosis
c. Goodpasture syndrome
d. Wegener granulomatosis

655. All of the following findings can be seen in the histopathological picture of a bronchial biopsy of a patient with respiratory insufficiency due to Asthma except



a. Smooth muscle hypertrophy
b. Basement membrane thickening
c. Eosinophilia
d. Squamous metaplasia

656. A 60-year-old mason complains of shortness of breath, which has become progressively worse during the past year. A chest X-ray shows small nodular shadows in both lungs. Pulmonary function studies reveal a pattern consistent with restrictive-lung disease. The patient subsequently develops congestive heart failure and expires. Autopsy discloses numerous small, fibrotic nodules in both lungs. Histologic examination of thesenodules is shown in the image. Which of the following is the most likely diagnosis?



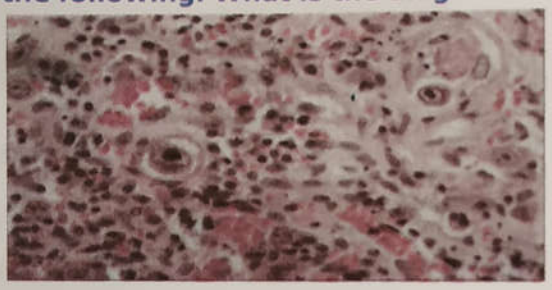
a. Anthracosis
b. Asbestosis
c. Sarcoidosis
d. Silicosis

653. c	654. a	655. d	656. d

657. What is true about Succinate dehydrogenase deficient GIST? (PGI)

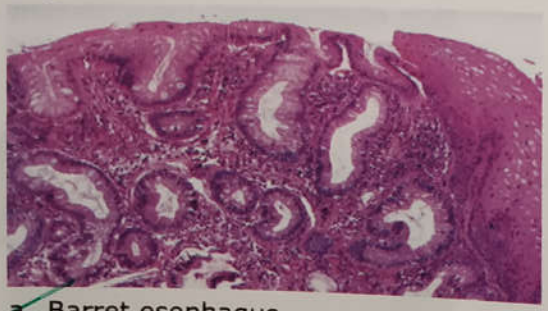
a. Negative for C-KIT
b. Younger age
c. Most common site is stomach
d. Sensitive to Imatinib
e. Aggressive clinical course

658. 40-years-old immunocompromized patient presented with complaints of dysphagia. UGI scopy showed multiple ulcers in the distal esophagus. Biopsy from the esophagus showed the following. What is the diagnosis?



a. Candida
b. Cytomegalovirus
c. Herpes viral infection
d. Eosinophilic esophagitis

659. A 40-years-old patient presented with heart burn and increased salivation. UGE scopy was done and biopsy was taken and is as shown below. Which of the following is the diagnosis?



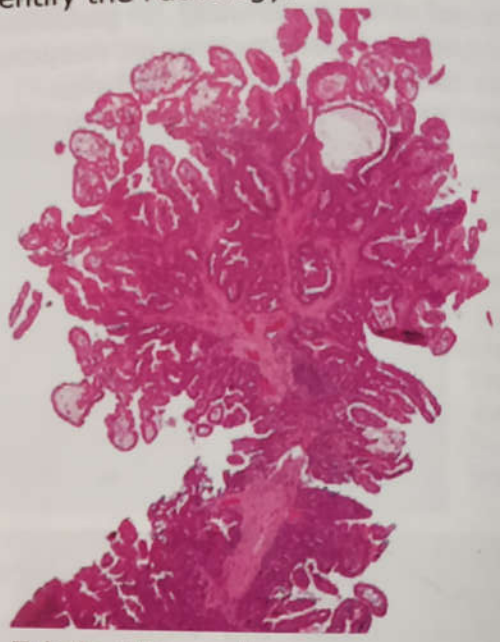
a. Barret esophagus
b. Adenocarcinoma
c. Esophagitis
d. Squamous cell carcinoma

660. Which of the following is associated with PAS positive macrophages?

a. Whipple disease
b. Abetalipoproteinemia
c. Crohn disease
d. Ulcerative colitis

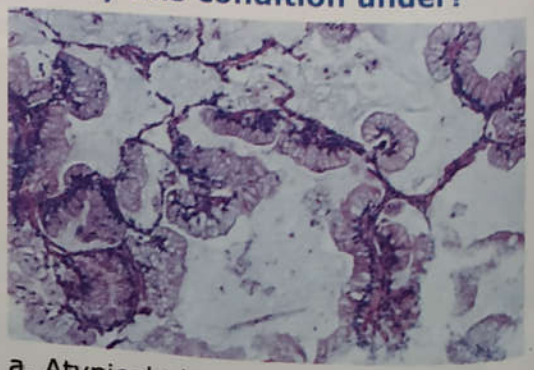
661. Pathologist examines biopsy from a patient presenting with bleeding per rectum with a past history of intussuption for the past 6 months. Histopathology obtained has been shown below.

Identify the Pathology?



a. Tubulo villous adenoma
b. Adenocarcinoma
c. Hamartomatous polyp
d. Juvenile polyposis syndrome

662. The following is a histopathological picture of a lung biopsy from a patient. Which category would you classify this condition under?



a. Atypicaladenomatoushyperplasia
b. Adenocarcinoma in situ
c. Minimally invasive adenocarcinoma
d. Invasive adenocarcinoma

657. a,b,c	658. b	659. a	660. a	661. c	662. b

663. Which of the following is a feature of Crohn's disease?

a. Pseudopolyps can be seen
b. Non-caseating granulomas are present
c. Backwash ileitis may be associated with Crohn's disease
d. Both b & c

664. Which of the following is the earliest change in intestine which occurs in Crohn's disease?

a. Cobblestone appearance
b. Aphthous ulcer
c. Perforation
d. Stricture

665. Which gene involved in colonic adenoma to colonic carcinoma?

a. P53
b. Rb
c. K RAS
d. EGFR

666. All of the following conditions are characterized by neoplastic polyps except-

a. Peutz Jeghers syndrome
b. Gardner syndrome
c. Turcot syndrome
d. Lynch syndrome

667. Mallory bodies contain-

a. Vimentin
b. Cytokeratin
c. Desmin
d. Collagen

668. All of the following drug induced liver injuries are correctly matched with their causative agents except

a. Valproate: Fatty liver disease
b. Amiodarone: Vascular lesions
c. Allopurinol: Fibrin ring granuloma
d. Angiosarcoma: Vinyl chloride

669. The pathway activated in inflammatory type of hepatocellular adenoma is

a. HNF1 alpha
b. WNT/Beta catenin
c. JAK/STAT
d. MAPK

670. Menetrier disease is caused by excessive secretion of

a. Transforming growth factor alpha
b. Tumor necrosis factor alpha
c. Transforming growth factor beta
d. IL-1

671. Which of the following gastric polyps are seen associated with familial adenomatosis polyposis?

a. Inflammatory
b. Hyperplastic
c. Fundic gland
d. Adenomatous

672. Most specific marker for GIST

a. ckit
b. CD 117
c. DOG1
d. Desmin

673. Microdeletion leading to instability of DNA, causing the cell to undergo degeneration is seen as a marker in which cancer?

a. Colon cancer
b. Breast cancer
c. Small cell lung cancer
d. Melanoma

674. Michaelis Gutmann bodies are seen in-

a. Xanthogranulomatous pyelonephritis
b. Malakoplakia
c. Nail patella syndrome
d. Chronic Pyelonephritis

675. Perivenular fibrosis is caused by:

a. Methotrexate
b. Alcohol
c. OCPs
d. Amiodarone

676. Hallmark of chronic hepatitis:

a. Interface hepatitis
b. Ballooning degeneration of hepatocytes
c. Cholestasis
d. Periportal fibrosis and bridging fibrosis

663. b	664. b	665. a	666. a	667. b	668. b	669. b	670. a	671. d
672. c	673. a	674. b	675. b	676. d				

677. A 1 month old child with conjugated billirubinemia and intrahepatic cholestasis. On Liver biopsy and staining with PAS red coloured granules were seen inside the hepatocytes. Probable diagnosis is?

a. Alpha 1 Antitrypsin deficiency
b. Congenital hepatic fibrosis
c. Wilson disease
d. Hereditary hemochromatosis

678. Most common gene responsible for hereditary hemochromatosis is?

a. HIV gene
b. HAMP gene
c. TfR2 gene
d. HFE gene

679. Most common gene associated with pancreatic cancer:

a. KRAS
b. SMAD
c. P53
d. Rb

680. Which of the following dyads are correctly matched regarding urinary casts & associated condition? (PGI)

a. Hyaline casts – may be normally present in healthy person
b. Muddy brown casts-acute tubular necrosis
c. WBC cast – pyelonephritis
d. Epithelial cast – acute glomerulonephritis
e. Myoglobin cast – Rhabdomyolysis

681. Seen in post streptococcal glomerulonephritis (PSGN)?

a. Nephrotic range proteinuria
b. Neutrophilic infiltration of tubules
c. Subepithelial deposits
d. Linear deposits along glomerular basement membrane

682. Most common renal lesions in HIV:

a. MPGN
b. RPGN
c. FSGS
d. Membranous nephropathy

683. Which is seen in Electron microscopy in PSGN?

a. Subpithelial humps
b. Spike and dome appearance
c. Mesangial deposits
d. Subendothelial deposits

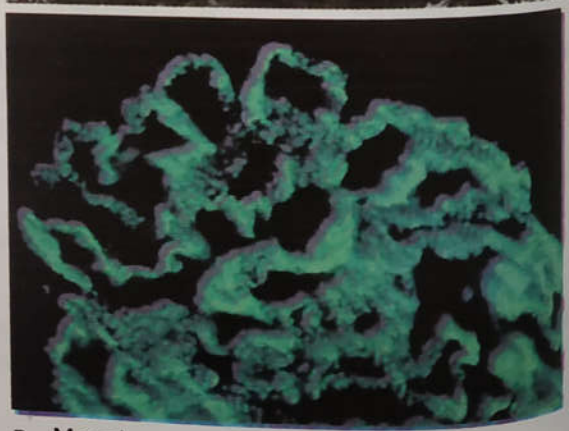
684. PSGN is associated with-(PGI)

a. Subepithelial deposits
b. Nephritis along with acute Renal failure rarely
c. Transiently low complement levels
d. HTN and proteinuria
e. Normal complements

685. Increased levels of C3NeF are associated with?

a. Type I MPGN
b. Type II MPGN
c. FSGS
d. Berger Disease

686. A 60-years-old male with complaints for frothy urine and facial puffiness. The DIF and electron microscopic picture of the patient is given below. Diagnosis is?



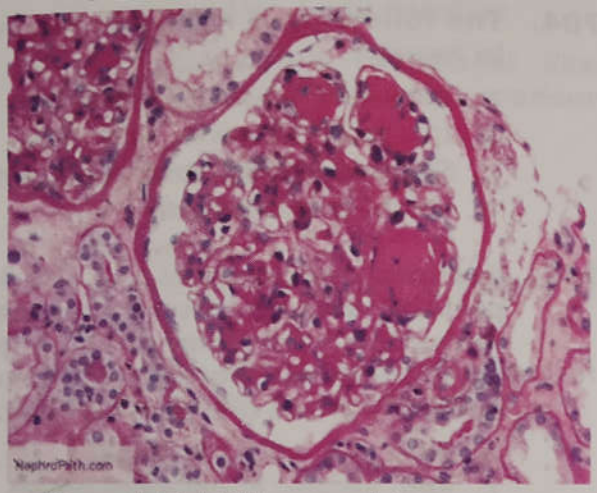
a. Membranoproliferative nephritis
b. Membranous glomerulopathy
c. Minimal change disease
d. FSGS

677. a	678. d	679. a	680. a,b,c,e	681. c	682. c	683. a
684. a,b,c,d	685. b	686. b				

687. Collapsing variant of FSGS is seen in:

a. NSAIDs
b. Heroin abuse
c. HIV
d. CMV

688. A 50-years-old male presented with blurring of vision. Urine examination showed proteinuria. Fundus examination showed dot and blot haemorrhages, microaneurysm and cotton wool spots. Histopathology pic of kidney given below. Your diagnosis?



a. Kimmelstiel Wilson nodules
b. Crescents
c. Amyloid
d. Segmental sclerosis

689. The intra cytoplasmic vacuoles seen in the Armanni Epstein cell are rich in-

a. Na and K
b. Glycogen
c. Lipids
d. Ketones

690. A 40-years-old hypertensive male was admitted to the hospital with sudden onset of headache and altered sensorium. On Examination his Blood Pressure was observed to be 220/110mm Hg and the patient died four later. What is likely pathological finding in his kidneys-

a. Small kidney with granular surface
b. Small kidney with petechial hemorrhages
c. Large kidney with waxy appearance
d. Large kidney with granular surface

691. Most common gene associated with renal cell carcinoma is?

a. WT-1
b. BRCA-1
c. VHL
d. PATCH

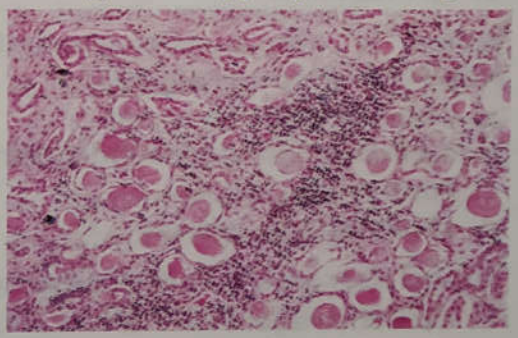
692. Chromophobe variant of renal cell carcinoma is associated with:

a. VHL gene mutations
b. Trisomy 7 and 17
c. 3 p deletions
d. Monosomy of 1 and Y

693. Cylindrical dilations of renal tubules is seen in-

a. Autosomal Recessive polycystic kidney disease
b. Medullary cystic disease
c. Autosomal dominant polycystic kidney diasese
d. Lipoid nephrosis

694. A 50-year-old woman complains of severe headaches anddizziness. The patient has a history of repeated urinary tractinfections. The blood pressure is 180/110 mm Hg. Laboratorystudies show elevated levels of BUN (38 mg/dL) and creatinine(2.8 mg/dL). A CT scan of the lower abdomen reveals small,irregularly shaped kidneys with deep coarse scars. A percutaneousrenal biopsy is shown. Which of the following is theappropriate diagnosis?



a. Acute pyelonephritis
b. Chronic pyelonephritis
c. Nephrosclerosis
d. Renal amyloidosis

695. Cleft like space in atheromatous plaque mainly contains?

a. Smooth muscle cell
b. Fibrous tissue
c. Cholesterol
d. Macrophages

687. c	688. a	689. b	690. b	691. c	692. d	693. a	694. b	695. c

696. Not true about monkeberg medial sclerosis:
a. Calcification of the walls of muscular arteries
b. Typically involving the internal elastic membrane
c. Persons older than age 50 are most commonly affected
d. Calcifications cause significant narrowing of vessel lumen

697. Characteristic histological finding in benign hypertension:
a. Proliferative end arteritis
b. Necrotizing arteriolitis
c. Hyaline arteriosclerosis
d. Cystic medial necrosis

698. Which of the following is seen in kidney in malignant hypertension?
a. Hyaline necrosis
b. Fibrinoid necrosis
c. Medial wall hyperplasia
d. Microaneurysm

699. Small vessel vasculitis is/are? (PGI)
a. Anti GBM ab
b. Takayasu arteritis
c. Kawasaki disease
d. Carcinoma induced vasculitis
e. IgA vasculitis

700. Which is not a characteristic of wegeners granulomatosis:
a. Granuloma in vessel wall
b. Focal necrotising glomerulonephritis
c. Positive for cANCA
d. Involves large vessels

701. Kaposi sarcoma all are true except?
a. Causative agent HHV 8
b. Can be Seen in depressed cell mediated immunity
c. Always associated with HIV
d. Vascular tumor

702. CD marker of angiosarcoma is?
a. CD 10 b. CD 19
c. CD 25 d. CD 31

703. Characteristic pathological finding in carcinoid of heart:
a. Fibrous endocardial thickening of right ventricle and tricuspid valve
b. Collagen deposition in wall of right ventricle
c. Interstitial fibrous thickening of right ventricles
d. Mononuclear inflammatory infiltrate in the wall

704. The following is not an aging change in heart
a. Increased LA cavity size
b. Increased LV cavity size
c. Sigmoid-shaped ventricular septum
d. Lambl excrescences

705. Autopsy specimen of the heart of a patient who diet due to myocardial infraction was stained with triphenyltetrazolium chloride dye. Colour of normal part of the heart will be:-
a. Blue b. White
c. Red d. Dark brown

706. In Rheumatic carditis, Mac Callum patch is seen in subendothelium of?
a. Right atrium
b. Right Ventricle
c. Left atrium
d. Left ventricle

707. Tigered effect in myocardium is due to:
a. Malignant change
b. Fatty change in heart
c. Seen in rheumatic fever
d. Associated with myocarditis

708. Which of the following breast cancer is bilateral usually?
a. Medullary b. Lobular
c. Mucinous d. Apocrine

709. Most common histological subtype associated with BRCA1 mutations is
a. Inflammatory b. Lobular
c. Papillary d. Medullary

696. d	697. c	698. b	699. a,d,e	700. d	701. c	702. d	703. a
704. b	705. c	706. c	707. b	708. b	709. d		

710. Highest susceptibility of breast cancer is seen with which gene mutation?

a. P53
b. BRCA1
c. BRCA2
d. Her 2 neu

711. BRCA2 mutated breast cancers show the following molecular profile

a. ER positive Her 2 neu positive
b. ER positive Her 2 neu negative
c. ER negative Her 2 neu positive
d. ER negative Her 2 neu negative

712. All are true about the basal like molecular group of breast cancer except

a. BRCA 1 mutation associated
b. Mainly grade I
c. Early relapse
d. Most common site of metastasis is bone

713. Which of the following is false about Her 2 neu positive breast cancers?

a. Usually low grade
b. Short timing of relapse
c. Can be apocrine or micropapillary in histology
d. P53 germline mutations associated

714. Most important prognostic factor for breast cancer without metastasis?

a. Tumor size
b. Lymph node status
c. ER/PR status
d. Her 2 neu status

715. Single file pattern of tumor cells with minimal desmoplasia is characteristic of which breast cancer?

a. Medullary
b. Lobular
c. Inflammatory
d. Papillary

716. Involvement of dermal lymphatics is seen in which subtype of breast cancer

a. Paget's disease
b. Inflammatory
c. Medullary
d. Lobular

717. Which of the following benign breast lesions has the most risk of malignant transformation?

a. Epithelial hyperplasia
b. Fibrocystic disease
c. Atypical ductal hyperplasia
d. Sclerosing adenosis

718. Migratory thrombophlebitis seen with all except

a. Carcinoma Breast
b. Carcinoma Colon
c. Carcinoma Prostate
d. Carcinoma Lung

719. Spongiform neurons are seen in-

a. Creutzfeldt-Jakob disease
b. Subacute sclerosing panencephalitis
c. Fatal familial insomnia
d. Cerebral toxoplasmosis

720. Verocay bodies are seen in-

a. Meningioma
b. Glioma
c. Medulloblastoma
d. Schwannoma

721. Which of the following is a DNA repair defect?

a. Bloom syndrome
b. Incontinentiapigmenti
c. Aplastic Anemia
d. Tuberous sclerosis

722. Childhood polycystic disease is -

a. Autosomal dominant
b. Autosomal recessive
c. X-linked
d. Multifactorial genetic disease

723. Marker for Squamous cell carcinoma

a. Vimentin
b. Cytokeratin
c. Desmin
d. Myogenin

724. SYT-SSX1 gene is associated with-

a. Liposarcoma
b. Rhabdomyosarcoma
c. Synovial sarcoma
d. Ewings sarcoma

710. b	711. b	712. b	713. a	714. b	715. b	716. b	717. c	718. c	719. a
720. d	721. a	722. b	723. b	724. c					

725. A glomerulus-like structure composed of central blood vessel enveloped by tumor cells within a space lined by tumor cells, is seen in-

a. Sertoli-Leydig cell tumor
b. Granulosa cell tumor
c. Endodermal sinus tumor
d. Sex cord tumor with annular tubules

726. Small cell cancer commonly metastasizes to-

a. Brain
b. Liver
c. Bone
d. Kidney

727. Most common cause of nephrotic syndrome in elderly

a. Membranous glomerulopathy
b. Minimal change disease
c. Acute GN
d. Focal GN

728. Perivascular lymphocytes and microglial nodules are seen in-

a. Multiple sclerosis
b. HIV encephalitis
c. CMV meningitis
d. Bacterial meningitis

729. Most common cerebellar tumor in children-

a. Astrocytoma
b. Medulloblastoma
c. Ependymoma
d. PNET

730. Tadpole cells or comma shaped cells on histopathology are seen in-

a. Trichoepithelioma
b. Seminoma
c. Rhabdomyosarcoma
d. Histiocytoma

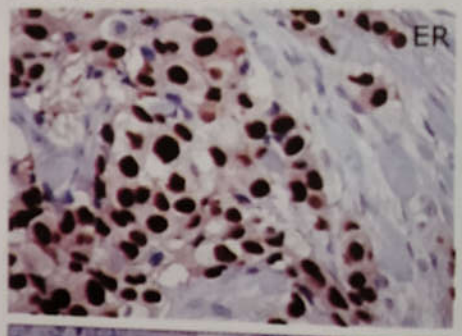
731. Maximum malignant potential is of which of the following conditions?

a. Familial adenomatous polyposis
b. Juvenile polyp
c. PeutzJegher polyp
d. Ulcerative colitis

732. A patient with history of smoking presents with a small <1 cm endobronchial nodule which was biopsied and studied. Electron microscopy showed the following. Which marker on immunohistochemistry would help reach a diagnosis?

a. TTF1
b. Napsin A
c. Synaptophysin
d. Calretinin

733. A 58 year old woman with breast cancer is being evaluated for further management. On IHC the following findings were obtained. Which of the following is true about the course of this patient's condition?



a. Trastuzumab is a treatment option
b. Hormonal therapy is not a treatment option
c. Patient has favourable prognosis
d. Patient falls into Basal subgroup of breast cancers

725. c	726. a	727. a	728. b	729. a	730. c	731. a	732. c	733. c

734. An 8 year old male patient with history of hematuria 3 days after contracting a respiratory infection is being evaluated. Which of the following is most likely to be the electron microscopy finding?

a. Basket weave pattern of the basement membrane
b. Subepithelial deposits
c. Mesangial deposits
d. Subendothelial deposits

735. All of the following genes are mutated in lung carcinoma except

a. EGFR b. KRAS
c. P53 d. ALK

736. Lymphoid aggregates in the form of follicles are seen in which of the following?

a. Hepatitis A b. Hepatitis B
c. Hepatitis C d. Hepatitis D

737. Following is a picture of a lung tissue biopsy from an elderly patient with smoking history and history of working in a shipyard. Which is the most common tumor that can occur in this patient?

a. Mesothelioma
b. Lung carcinoma
c. Laryngeal carcinoma
d. Nasopharyngeal carcinoma

738. All of the following are characteristic features of malignancy except

a. Atypical mitosis
b. Hyperchromatic nuclei
c. Loss of polarity
d. Chromosomal diploidy

739. All of the following features can help differentiate Crohn's disease from Ulcerative colitis except

a. Granuloma formation
b. Cryptab scesses
c. Transmural inflammation
d. Pseudopoylps

740. Lardaceous spleen occurs due to

a. Deposition of amyloid in white pulp
b. Deposition of amyloid in red pulp
c. Deposition of hemosiderin
d. Deposition of calcium

741. Tumor marker(s) associated with both colon cancer and pancreatic cancer is

a. CEA b. CA-125
c. CA 15-3 d. All of the above

742. Which of the following has the least chance of metastasising to the brain?

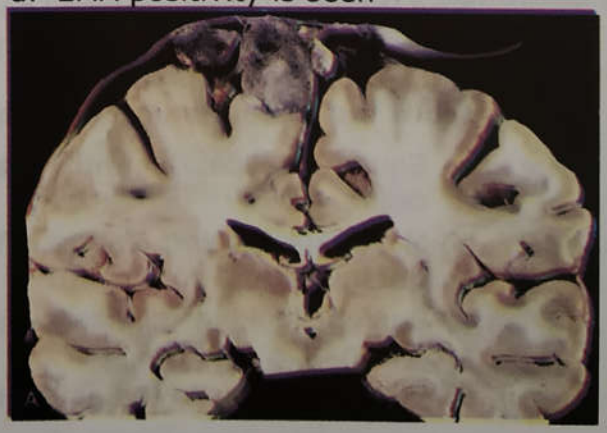
a. Lung carcinoma
b. Melanoma
c. Breast carcinoma
d. Prostate carcinoma

743. Coffee bean nuclei are seen in all except

a. Brenner tumor
b. Langerhan's cell histiocytosis
c. Follicular carcinoma thyroid
d. Chondroblastoma

744. All of the following are true about the following tumor except

a. Can express progesterone receptor
b. Rhabdoid morphology is grade III
c. Bone invasion upgrades the tumor
d. EMA positivity is seen



734. c	735. d	736. c	737. b	738. d	739. b	740. b	741. a	742. d
743. c	744. c							

745. The following is triphenyl tetrazolium chloride stain of a post-mortem heart, the corresponding labelling to 1,2 3 are

a. Viable myocardium, recent infarct, old infarct
b. Old infarct, recent infarct, viable myocardium
c. Recent infarct, old infarct, viable myocardium
d. Pericardial fat, recent infarct, viable myocardium

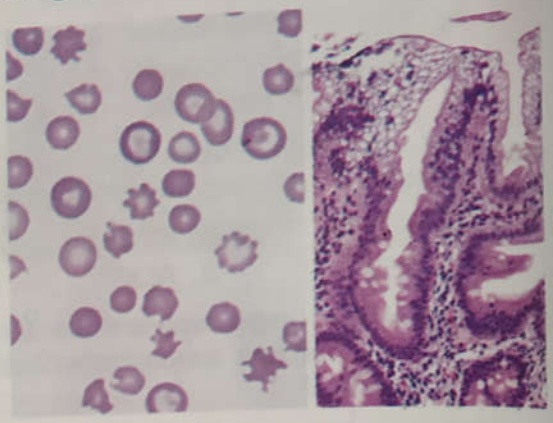
746. Not ture about microscopic polyangitis is

a. Lungs are rarely involved
b. Glomerulonephritis is frequent
c. Anti MPO anti bodies
d. Leucocytoclastic vasculitis

747. A 35-year old smoker presents with severe retro sternal pain which is radiating to neck and worsens after heavy meals occasionally it makes him wake up at night. Upper GI Endoscopic examination does not reveal any mass but a velvety patch in the lower esophagus. Biopsy from the lesion is likely to show

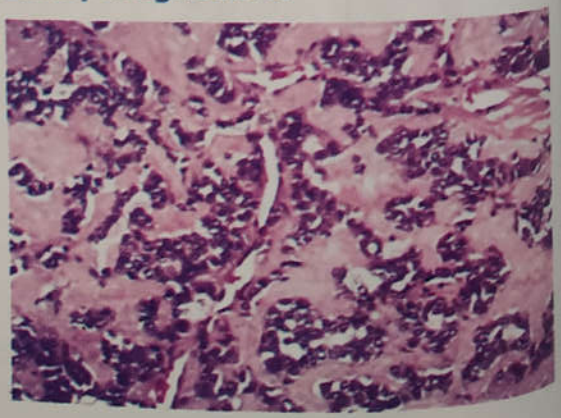
a. Squamous pearls with malignant cells
b. Malignant looking glands infiltrating the muscle
c. Benign looking glands which are Alcian blue positive
d. Viral inclusions in the epithelial cells

748. A 2-year old is having a long standing diarrhea and steatorrhea. The child is also short in stature and has not gained weight. The peripheral smear and an intestinal biopsy was done and is as shown in the figure. What is the likely diagnosis?



a. celiac disease
b. Whipples Disease
c. Abetalipoproteinemia
d. Lactase deficiency

749. A 7-year child has a mass in the front of neck. Notable findings are BP of 160/90 mm of Hg. His father also had history of hypertension and a neck surgery was performed on him Biopsy from the mass is as follows. Likely diagnosis is



a. Papillary carcinoma of thyroid
b. Parathyroid hyperplasia
c. Medullary carcinoma of thyroid
d. Carotid body tumor

745. c	746. a	747. c	748. c	749. c

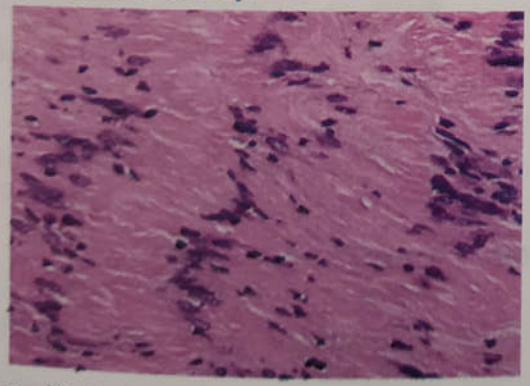
750. The following gross picture and histopathological picture is of which condition

a. Capillary hemangioma
b. Cavernous hemangioma
c. Lymphangiosarcoma
d. Kaposi's sarcoma

751. A portion of large bowel of a young patient is shown below. The patient most likely harbors a germline mutation in which of the following genes?

a. KRAS
b. p53
c. MYC
d. APC

752. All of the following are true about the following cerebellopontine angle tumor except



a. Well circumscribed mass
b. S-100 is negative
c. Associated with neurofibromatosis type 2
d. Areas of nuclear palisading are seen

753. All are features of Crohn's disease except

a. Earliest feature is aphthous ulcer
b. Elongated, serpentine ulcers oriented along the axis of the bowel
c. Intestinal wall is thinned out
d. Cobblestone appearance

754. Mallory Denk bodies are composed of

a. Cytokeratin 8/18
b. Cytokeratin 5/6
c. Vimentin
d. Cytokeratin 20

755. All of the following are true about familial breast cancers with BRCA1 gene mutations except

a. Luminal A subtype
b. Poorly differentiated
c. Medullary features
d. Seen in younger women

756. All of the following are features of a stable plaque except

a. Large lipid core
b. Less neutrophils
c. Thick fibrous cap
d. Smooth muscle cells

757. The rule of "10's" of phaeochromocytoma stands for all of the following except

a. Extra adrenal
b. Bilateral
c. Malignant
d. Familial

758. Most common pituitary adenoma in MEN-1 is

a. Lactotroph
b. Somatotroph
c. Corticotroph
d. Gonadotroph

759. All of the following are epithelial tumors of stomach except

a. Signet ring cell carcinoma
b. Neuroendocrine tumor
c. Granular cell tumor
d. Undifferentiated carcinoma

750. a	751. d	752. b	753. c	754. a	755. a	756. a	757. d	758. a	759. c

760. Which of the following is false about the most common primary cardiac tumor in adults?

a. Most common site is left atrium
b. Associated with Carney triad
c. Activating mutations in GNAS
d. Wrecking ball effect

761. A patient with diffuse enlargement of thyroid gland showed the following histopathological picture. Which of the following statements is false about this condition?

a. CD4 mediated killing of epithelial cells
b. Female preponderance
c. Thyrotoxicosis may occur
d. Most common cancer arising in this condition is papillary carcinoma

762. The following gross image of liver shows

a. Metastasis
b. Focal nodular hyperplasia
c. Leiomyoma
d. Hepatic adenoma

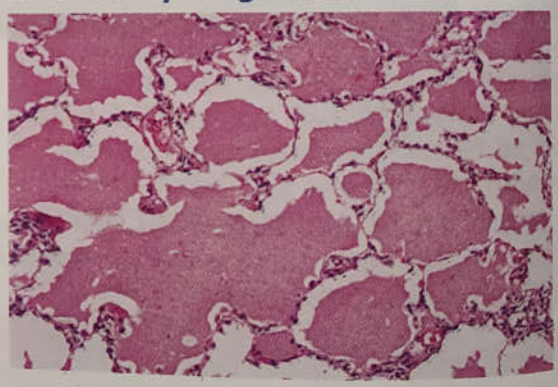
763. Mutations associated with nonsmall cell Lung ca are? (PGI)

a. p53
b. EGFR
c. Rb
d. Myc
e. RAS

764. Hyaline membrane in the lung is seen in-

a. Respiratory distress syndrome
b. Pulmonary edema
c. Pneumococcal pneumonia
d. Acute viral hepatitis

765. A 22-year-old man who is being treated for leukemia complains of shortness of breath on exertion, pleuritic chest pain, and a low-grade fever. Physical examination reveals crackles in both lung bases and clubbing of the figers. Bronchoalveolar lavage demonstrates PAS-positive material and elevated levels of surfactant proteins. An open-lung biopsy is shown in the image. Which of the following is the most likely diagnosis?



a. Alveolar proteinosis
b. Eosinophilic pneumonia
c. Goodpasture syndrome
d. Hyaline membrane disease

766. Morphologic features of celiac disease include all, except:

a. Increase in intra-epithelial lymphocytes
b. Increase crypt : villous ratio
c. Distended macrophages with PAS positive granules in lamina propria
d. Elongated, hyperplastic and tortuous crypts

760. b	761. a	762. a	763. a, b ,e	764. a	765. a	766. b

767. In carcinoma of unknown primary, if the tissue marker CDX-2 is positive, it indicates:

a. Bladder cancer
b. Gastrointestinal cancer
c. Lung cancer
d. Thyroid cancer

768. Councilman bodies are seen in-

a. Alcoholic cirrhosis
b. Wilson's disease
c. Acute viral hepatitis
d. Autoimmune hepatitis

769. Piecemeal necrosis on liver biopsy is a feature of-

a. Alcoholic hepatitis
b. Indian childhood cirrhosis
c. Chronic active hepatitis
d. Primary alcoholic cirrhosis

770. Chromosome involved in Wilson's disease

a. 11 b. 12
c. 13 c. 14

771. Giant hypertrophy of gastric mucosa similar to brain surface is seen in-

a. Peptic cell metaplasia
b. Carcinoma stomach
c. Menetrier's disease
d. Leiomysarcoma

772. Gastric carcinoma is associated with-(PGI)

a. Inactivation of p53
b. Over expression of C-erb-B2
c. Over expression of C-MET
d. Activation of RAS

773. Skip lesions of colon with epithelioid granuloma are usually seen with-

a. Crohn's disease
b. Ulcerative colitis
c. Intestinal TB
d. Sarcoidosis

774. Gluten sensitive enteropathy is most strongly associated with-

a. HLA-DQ2 b. HLA-DR4
c. HLA-DQ3 d. HLA DR2

775. Antigliadin antibodies are detectable in-

a. Tropical sprue
b. Whipple's disease
c. Celiac disease
d. Intestinal lymphoma

776. Massive hepatocellular necrosis is seen with:

a. Tetracycline
b. Macrolides
c. Methyldopa
d. Acetaminophen

777. Fibrin ring granuloma in liver is caused by:

a. Suphonamides
b. Amlodarone
c. Isoniazid
d. Allopurinol

778. Peliosis hepatis is caused by:

a. Contraceptives
b. Anabolic steroids
c. Erythromycin
d. Ezetemibe

779. Macrovesicular fatty change in hepatocytes is seen due to infection with:

a. Hepatitis A
b. Hepatitis B
c. Hepatitis C
d. Hepatitis D

780. Microvesicular fatty liver is caused by:

a. DM
b. Valproate
c. Starvation
d. IBD

781. On kidney biopsy, PAS positive structures are:

Glomerular basement membrane

a. Tubule
b. Neutrophils
c. Interstitium
d. Mesangial matrix

767. b	768. c	769. c	770. c	771. c	772. a	773. a	774. a	775. c	776. d
777. d	778. b	779. c	780. b	781. c,d					

782. Rapid advanced renal parameters is clinically termed as Rapidly progressive glomerulonephritis. What exactly is the finding on microscopy?

a. Membrane thickening
b. Segmental sclerosis
c. Crescents
d. Mesangial expansion

783. True about membranoproliferative glomerulonephritis is? (PGI)

a. Low C3 level
b. Double basement membrane appearance on light microscopy
c. Dense deposit along basement membrane on electron microscopy
d. Mesangial hypocellularity
e. GBM thickening

784. Which of the following disease has deafness as well as hematuria?

a. Alport Syndrome
b. Good pasture syndrome
c. IgA nephropathy
d. Cryoglobinemia

785. In renal biopsy of a 14 years old boy with nephritic syndrome, glomeruli are showing proliferation of mesangial cells with GBM thickening and mesangial cell interposition. What is the most likely diagnosis in this case?

a. Membranous nephropathy
b. Diffuse proliferative glomerulonephritis
c. Focal segmental glomerulosclerosis
d. Mesangiocapillary glomerulonepthritis

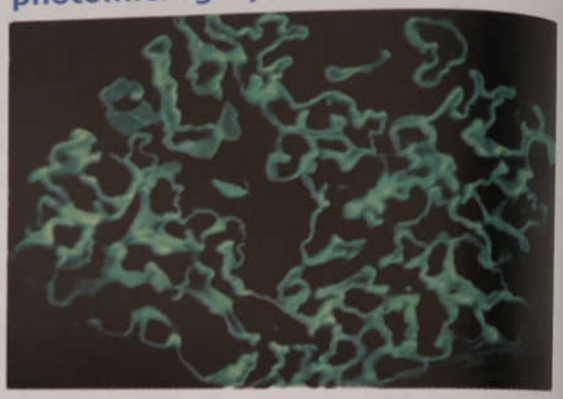
786. Dysmorphic RBC with ARF is seen in?

a. Glomerular disease
b. Renal carcinoma
c. Proximal tubule disease
d. Distal tubule disease

787. Mesangial cells of IgA Nephropathy overexpresses:

a. CD51
b. CD61
c. CD71
d. CD81

788. A 35-year-old man with a history of smoking presents with hematuria and bloody sputum. Over the next 2 days, he develops oliguria and renal failure, after which he is placed on dialysis. A renal biopsy is stained with fluorescein-conjugated goat antihuman IgG, and the results are shown. Which of the following best describes the pattern of direct immunofluorescence observed on this photomicrograph?



a. Discontinuous and peripheral
b. Finely granular along the perimesangial reflections
c. Linear along the glomerular basement membrane
d. Mesangial with a stalk predominance

789. Berger nephropathy is due to mesangial deposition of-

a. Fibrin and C3
b. IgD and C3
c. IgE and C3
d. IgA and C3

790. In IgA nephropathy (Berger's disease) there are-

a. Subepithelial deposits
b. Subendothelial deposits
c. Mesangial deposits
d. Basement membrane deposits

791. Kimmelstiel-Wilson lesions in kidney consists-

a. Splitting of glomerular basement membrane
b. Nodular sclerosis of the glomeruli
c. Hyaline sclerosis
d. Hyperplastic arteriosclerosis

782. c	783. a,b,c,e	784. a	785. d	786. a	787. c	788. c	789. d	790. c	791. b

792. Pauci immune glomerulonephritis is seen in:

a. After transplant in Alports
b. Microscopic polyangitis
c. Henoch-schonlein nephritis
d. Lupus nephritis

793. Mutation in NPHS1 gene causes which disease?

a. Alport syndrome
b. Congenital Finnish type nephrotic syndrome
c. Focal segmental glomerulosclerosis
d. Nail patella syndrome

794. Broad cast is characteristic of-

a. CRF
b. Acute glomerulonephritis
c. ARF
d. Renal TB

795. Cast is produced due to damage to-

a. Nephron b. Tubule
c. PCT d. DCT

796. Maximum endocapillary proliferation is a feature of-

a. Membranous GN
b. Mesangioproliferative
c. Focal glomerulosclerosis
d. Post-streptococcal GN

797. Rapidly progressive glomerulonephritis is histologically characterised by the presence of numerous-

a. Intramembranous dense depositis
b. Atrophic proximal convoluted tubules
c. Hyalinized small arterioles
d. Epithelial cell crescents

798. Crescent are derived from-

a. Parietal epithelial cells + fibrin + macrophage
b. Mesangium + fibrin + macrophage
c. Tubule + mesangium + fibrin
d. Mesangium + fibrin

799. Malignancies are most commonly associated with which type ofGlomerulonephritis?

a. MGN
b. MPGN
c. Minimal change disease
d. FSGS

800. Most common cause of death in APCKD (Adult Polycystic Kidney Disease)-

a. Chronic renal failure
b. Cardiovascular disease
c. Ruptured berry aneurysm
d. Sepsis

801. The mother of a 2-month-old child palpates a mass on the left side of the child's abdomen. Vital signs are normal. A CT-guided renal biopsy shows undifferentiated tubules surrounded by undifferentiated mesenchyme, smooth muscle, and islands of cartilage. The mass is removed (shown in the image) and displays variably sized cysts. Which of the following is the most likely diagnosis for this child's flank mass

a. Autosomal recessive polycystic kidney disease
b. Autosomal dominant polycystic kidney disease
c. Medullary sponge kidney
d. Renal dysplasia

792. b	793. b	794. a	795. b	796. d	797. d	798. a	799. b	800. b	801. d

802. An 8-year-old boy presents with headaches, dizziness, and malaise approximately 2 weeks after a severe sore throat. His mother describes puffiness of his face and darkening of his urine. She also notes that her son is passing less urine and that he is becoming increasingly short of breath. On physical examination, there is anasarca, hypertension (190/130 mm Hg), and tachycardia. The urine is scanty and brownish red. Urinalysis shows 3+ proteinuria. Microscopic examination of the urine discloses numerous RBCs, as well as occasional granular and red cell casts. A renal biopsy is stained by direct immunofluorescence microscopy for complement C3, and the results are shown. Which of the following best describes the pattern of immune-fluorescence observed in this renal biopsy?

a. Deposits limited to the mesangium
b. Granular deposits along the perimesangial reflections
c. Linear staining along the glomerular basement membranes
d. Subepithelial and subendothelial deposits

803. In Wegener's granulomatosis cytoplasmic anti neutrophilic antibodies are directed against:

a. Proteinase 1
b. Proteinase 2
c. Proteinase 3
d. Proteinase 4

804. Type of renal cell carcinoma seen in sickle cell trait is

a. Chromophobe
b. Clear cell
c. Papillary
d. Medullary

805. Which one is found in HSP?

a. IgA
b. IgM
c. IgG
d. IgE

806. Arterial biopsy of elderly male show fragmentation of elastic lamina, lymphocyte infiltration and giant cells-

a. Temporal arteritis
b. Takayasu disease
c. Polyarteritisnodosa
d. Kawasaki disease

807. Silk Road disease is:

a. Behcet's syndrome
b. Giant cell arteritis
c. Henoch schonlein purpura
d. Wegener's granulomatosis

808. Necrotizing arterioritis with fibrinoid necrosis is:

a. Immediate hypersensitivity
b. Cell mediated immunity
c. Antigen-antibody complex mediated
d. Cytotoxic cell mediated

809. A 55-years-old male presents with severe chest pain radiating to the left arm. ECG shows ST segment elevation in the V4, V5 and V6 leads. CK-MB and troponin levels are found to be increased. The most likely cause for the increase in enzyme in serum is:

a. Clumping of nuclear chromatin
b. Lysosomal Autophagy
c. Mitochondrial swelling
d. Cell membrane defects

810. Which of the following salivary gland tumors have a variable mixture of three types of cells?

a. Pleomorphic adenoma
b. Mucoepidermoid carcinoma
c. Warthin's tumor
d. Adenoid cystic carcinoma

802. b	803. c	804. d	805. a	806. a	807. b	808. c	809. d	810. d

811. Irreversible change in ischemia of heart occurs in?
a. 10 min
b. 30 min
c. 60 min
d. 90 min

812. Post mortem finding in a case of death due to myocardial infarction is?
a. Fat necrosis
b. Caseous necrosis
c. Liquefactive necrosis
d. Coagulative necrosis

813. Which of the following is not true about medullary carcinoma of breast?
a. Relatively good prognosis as compared to other poorly differentiated tumors
b. Rich lymphocytic infiltrates
c. Associated with BRCA2 mutations
d. Maximum desmoplasia

814. Which of the following benign breast lesions is non proliferative
a. Ductal papilloma
b. DCIS
c. Sclerosing adenosis
d. Apocrine metaplasia

815. Patients with Hashimoto's thyroiditis are at increased risk of developing-
a. Papillary carcinoma
b. Follicular carcinoma
c. T-cell lymphoma
d. B-cell lymphoma

816. Paraneoplastic syndromes are classically associated with (PGI type)
a. Renal ca
b. Oesophagealca
c. Breast ca
d. Testicular ca
e. Small cell carcinoma of lung

817. Which of the following soft tissue tumors is not caused by fusion of EWS with another gene?
a. Ewing sarcoma family tumors
b. Extraskeletal myxoid chondrosarcoma
c. Desmoplastic small round cell tumor
d. Alveolar soft part sarcoma

818. Ectopic ADH production is seen in-
a. Small cell carcinoma is lung
b. Anaplastic carcinoma of lung
c. Squamous cell carcinoma of lung
d. Adenocarcinoma of cerebellum

819. Retinoblastoma is associated with-
a. Osteosarcoma
b. Hepatocellular carcinoma
c. Squamouscell carcinoma
d. Osteoclastoma

820. Most common cancer arising in a patient with chronic osteomyelitis is
a. Squamous cell carcinoma
b. Osteosarcoma
c. Fibrosarcoma
d. Osteoid osteoma

821. High risk of malignancy is seen in?
a. Simple hyperplasia with atypia
b. Simple hyperplasia without atypia
c. Complex hyperplasia with atypia
d. Complex hyperplasia without atypia

822. S 100 is a marker of?
a. Melanoma
b. Schwannoma
c. Histiocytoma
d. All of the above

823. N-MYC amplification is associated with which tumor?
a. Burkitt lymphoma
b. Squamous cell carcinoma lung
c. Astrocytoma
d. Neuroblastoma

824. Pseudomyxoma peritonei is seen with -
a. Mucinous cystadenocarcinoma ovary
b. Carcinoidappedix
c. Endometrial carcinoma
d. Ileal carcinoid

811. b	812. d	813. d	814. d	815. d	816. a,e	817. d	818. a	819. a	820. a
821. c	822. d	823. d	824. a						

825. A child is born with a single functional copy of a tumor suppressor gene. At the age of 5 years, the remaining normal allele is lost through mutation. As a result, the ability to control the transition from G1 to the S phase of the cell cycle is lost. Which of the following neoplasms is most likely to arise by means of this mechanism-
a. Retinoblastoma
b. Breast carcinoma
c. Adenocarcinoma of colon
d. Cerebral astrocytoma

826. Keratin is a tumour marker for-
a. Carcinoma cervix
b. Neurofibroma
c. Rhabdoyosarcoma
d. Choriocarcinoma

827. Content of Aschoff's nodules are A/E
a. Histiocytes
b. Langhan's giant cell
c. Fibrinoid deposition
d. Aschoff's cells

828. PTH like substance is produced by which type of lung malignacy-
a. Squamouscell carcinoma
b. Oatcell carcinoma
c. Adeno carcinoma
d. Large cell carcinoma

829. Nephrotic syndrome is characterised by all except
a. Proteinuria b. Hyperlipidemia
c. Oedema d. Haematuria

830. Hypercoagulation in nephrotic syndrome is caused by-
a. Loss of antithrombin III
b. Decreased fibrinogen
c. Decreased metabolism of Vitamin K
d. Increase in protein C

831. Skip granulomatous lesions are seen in-
a. Ulcerative colitis
b. Crohn's disease
c. Whipple's disease
d. Reiter's disease

832. True about Barrett's esophagus is?
a. Squamous metaplasia
b. Columnar metaplasia
c. Irreversible
d. Not premalignant

833. The highest malignant potential is seen in-
a. Crohn's disease
b. Ulcerative colitis
c. FAP
d. PeutzJegher syndrome

834. Macrophages containing PAS positive granules and rod-shaped bacilli in small intestinal mucosa are found in-
a. Agammaglobulinemia
b. Tropical sprue
c. Whipple's disease
d. Coeliac spure

835. Psammoma bodies can be seen in the following except:
a. Follicular carcinoma of thyroid
b. Papillary carcinoma of thyroid
c. Meningioma
d. Serous cystadenoma of ovary

836. Myotonic dystrophy is inherited in chromosomes-
a. 21 b. 20
c. 19 d. 24

837. All are true about silicosis except?
a. Bifringent crystals seen
b. Pleural plaque may be seen
c. Lower lobe is usually involved
d. Most common pneumoconiosis

838. Asbestosis causes?
a. Lymphoma
b. Leukaemia
c. Renal cell carcinoma
d. Mesothelioma

839. In thyroglossal cyst, carcinoma which arises is-
a. Follicular b. Medullary
c. Papillary d. Anaplastic

825. a	826. a	827. b	828. a	829. d	830. a	831. b	832. b	833. c	834. c
835. a	836. c	837. c	838. d	839. c					

840. MEN-2 syndrome is associated with increase in-
a. Papillary Ca of thyroid
b. Medullary
c. Follicular
d. Anaplastic

841. Most common primary tumor of heart?
a. Myxoma
b. Rhabdomyosarcoma
c. Fibroma
d. Leiomyosarcoma

842. Which one of these thyroid cancers cannot be diagnosed by FNAC?
a. Papillary b. Medullary
c. Follicular d. Anaplastic

843. Pseudopolyps are features of-
a. Crohn's disease
b. Ulcerative colitis
c. Celiac sprue
d. Whipple's disease

844. Tumor marker for lung adenocarcinoma? (PGI)
a. CK 7
b. CK 20
c. TTF1
d. CK 5/6
e. BerEP- 4

845. Reid's index is-
a. Increased in chronic bronchitis
b. Decreased in chronic bronchitis
c. Increased in bronchial asthma
d. Decreased in bronchial asthma

846. True about light microscopy in minimal change disease is:
a. Loss of foot process seen
b. Anti GBM Abs seen
c. IgA deposits seen
d. No change seen

847. Chromosome associated with familial polyposis colon?
a. Chromosome 5
b. Chromosome 6
c. Chromosome 11
d. Chromosome 13

848. Buerger disease is associated with:
a. Alcoholism
b. Smoking
c. Trauma
d. Cold environment

849. Which of the following soft tissue tumors is not caused by fusion of EWS with another gene?
a. Ewing sarcoma family tumors
b. Extraskeletal myxoid chondrosarcoma
c. Desmoplastic small round cell tumor
d. Alveolar soft part sarcoma

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b. Hepatocellular carcinoma
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d. Osteoclastoma

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b. Osteosarcoma
c. Fibrosarcoma
d. Osteoid osteoma

853. High risk of malignancy is seen in?
a. Simple hyperplasia with atypia
b. Simple hyperplasia without atypia
c. Complex hyperplasia with atypia
d. Complex hyperplasia without atypia

854. Gene of Wilson's disease is-
a. ATP 7^a b. ATP 7B
c. ADP 7^a d. ADP 7B

855. Most common benign tumor of liver is
a. Adenoma
b. Hemangioma
c. Focal nodular hyperplasia
d. Hepatoblastoma

840. b	841. a	842. c	843. b	844. a,c,e	845. a	846. a	847. a	848. b	849. d
850. a	851. a	852. a	853. c	854. b	855. b				

856. Which of the following special stains specifically help to identify goblet cells in a case of suspected Barret's esophagus?

a. Alcian blue at pH 2.5
b. Mucicarcmine
c. PAS
d. Masson trichrome

857. Ulcerative colitis is characterised by all except

a. Mucosal and submucosal involvement
b. Absent granulomas
c. Brisk Lymphoid aggregates
d. Minimal edema

858. Early to late adenoma transformation in colorectal carcinoma is mediated by

a. Loss of APC
b. Activating mutations of KRAS
c. Loss of p53
d. Loss of SMAD4

859. Exfoliative cytology is useful in -

a. Carcinoma stomach
b. Carcinoma bronchus
c. Carcinoma cervix
d. All of the above

860. Atheromatous plaque do not contain:

a. Platelet
b. Neutrophils
c. Smooth muscle fibres
d. Monocytes

861. Autopsy finding after 12hrs in a case of death due to M.I.-

a. Caseous necrosis
b. Coagulative necrosis
c. Fat necrosis
d. Liquefactive necrosis

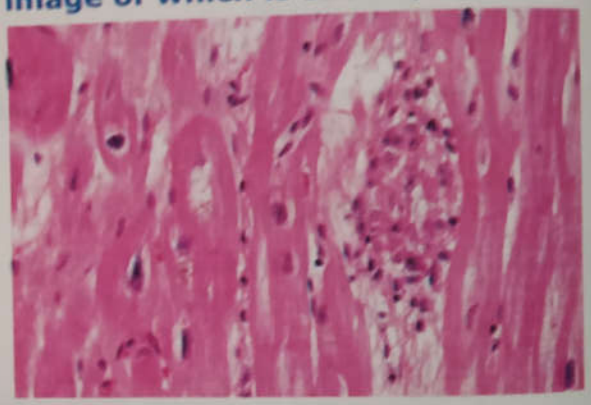
862. Nottingham system of grading breast cancers of IDC subtype does not include which of the following parameters

a. Necrosis
b. Mitotic activity
c. Nuclear pleomorphism
d. Tubular differentiation

863. True about Takayasu is all except?

a. Causes transmural granuloma
b. Granulomatous vasculitis
c. Large to medium vessel
d. Called giant cell aortoarteritis

864. 30-years-old mal presented with severe dyspnoea and fatigue. X-ray showed left atrial enlargement. Physician suspects the patient of having mitral stenosis and gets a histopath examination done, the image of which is shown, it shows?



a. Sarcoidosis
b. Tuberculosis
c. Aschoff bodies
d. Fungal granuloma

865. Anti transglutaminase antibody is seen in?

a. Celiac disease
b. SLE
c. Collagenous colitis
d. Tropical sprue

856. a	857. c	858. b	859. d	860. b	861. b	862. a	863. d	864. c	865. a

636. D

Gain of function mutations in RET leads to MEN 2 and Medullary carcinoma thyroid.

637. A

Option A >C - epithelial malignancy marker

Option B- mesenchymal malignancy marker

Option D- marker for lymphomas

638. C

Multinucleated giant cells with refractile matter can be seen in the picture suggestive of aspiration pneumonia.

639. A

The gross specimen shows dilated bronchioles close to the pleura.

Option B- localised mass with necrosis

Option C- infiltrate grey white mass

Option D- tiny grey while millet like areas spread across the entire lung

640. D

641. B

Features of silicosis

- Associated with sandblasting, foundries, mines.
- Macrophages respond to silica and release fibrogenic factors, leading to fibrosis.
- It is thought that silica may disrupt phagolysosomes and impair macrophages, increasing susceptibility to TB.
- Risk of cancer, cor pulmonale, and Caplan syndrome
- Affects upper lobes
- Egg shell calcification seen in hilar lymph nodes om CXR
- Collagenous silicotic nodules in the lung

Option CE are features of coal workers pneumoconiosis

642. A

In the new WHO classification, the number of subtypes has been reduced to three, which makes the diagnosis easier and avoids rare subtypes with confusing names

- Keratinizing
- Non-keratinizing
- Basaloid squamous cell carcinoma
- PreinvasivelesionSquamouscell carcinoma in situ

643. A, C, D, E

Option B- positive in both

Options AD- positive in adenocarcinoma

Option CE- positive in mesothelioma

644. C

Most malignant and aggressive form of lung cancer is small cell carcinoma

645. A, C, E

Option B- marker for primary lung carcinoma

Option D- marker for hepatocellular carcinoma

646. C

647. C

648. A

Most common paraneoplastic syndrome associated with small cell carcinoma is ectopic hormone production, ACTH and ADH being most common.

649. B

Option A- most common type of emphysema

650. C

Asbestos (ferruginous) bodies are golden-brown fusiform rods resembling dumbbells, found in alveolar sputum sample or histology, visualized using Prussian blue stain.

651. C

Tuberculosis can present with both caseating and non caseating granulomas. Bilateral hilar lymphadenopathy is characteristic of sarcoidosis.

652. C

An entire lobe shows consolidation hence C, Klebsiella being the probable cause.

653. C

Cup and saucer appearance on special silver stain with history of immunocompromise- Pneumocystis

654. A

Panacinar emphysema is seen with option A

Option B will show bronchiectasis

Option D may show cavitations

655. D

Option ABC are features of bronchial asthma

Option D is seen in chronic bronchitis.

656. D

Picture shows collagenous silicotic nodules suggestive of silicosis.

657. A, B, C

SDH deficient GIST is ckit negative and hence non responsive to Imatinib and they have an indolent course. It is a part of Carney Stratakis Syndrome.

658. B

Biopsy shows owl eye inclusions and the history is suggestive of CMV inclusions

Option A- pseudohyphae can be seen

Option C- multinucleation, margination and nuclear molding can be seen

Option D- increased number of eosinophils

659. A

The history and histology of transition from squamous to columnar shows Barret's metaplasia with goblet cells. Adenocarcinoma would show infiltrating glands of varying shapes and sizes.

660. A

Tropheryma whippeli, a gram positive Actinomycetes engulfed by macrophages give them a foamy appearance and they stain positive with PAS.

Option B- lipid laden epithelial cells

Option C- architectural distortion, cryptitis, aphthous ulcer, non caseating granulomas, transmural inflammation

Option D- cryptitis, superficial inflammation, pseudopolyps

661. C

Histology shows arborizing smooth muscle bundles with glands arising from them suggestive of PeutzJegherpolp which is a hamartomatous polyp.

662. B Adenocarcinoma in situ

Adenocarcinoma in situ is a pre invasive lesion depicted in this picture. Thecells may be columnar and mucus producing or cuboidal andsimilar to type II pneumocytes. They tend to grow along thealveolar septa, as depicted in the photomicrograph

663. B

A and C are features characteristic of ulcerative colitis.

664. B

The earliest lesion, the aphthous ulcer, may progress, and multiple lesions often coalesce into elongated, serpentine ulcers oriented along the axis of the bowel. Edema and loss of the normal mucosal texture are common. Sparing of interspersed mucosa, a result of the patchy distribution of Crohn disease, results in a coarsely textured, cobblestone appearance in which diseased tissue is depressed below the level of normal mucosa

665. A

KRAS mutation is involved in transformation of early adenoma to late adenoma.

666. A (single best option)

Although D is a non polyposis condition, it is neoplastic as it B and C. A is an example of hamartomatous polyps and hence is the best option here.

667. B

CK 8/18 skeins left after damage are called Mallory Hyaline bodies

Pattern of Injury	Morphologic Findings	Examples of Associated Agents
Cholestasis	Bland hepatocellular cholestasis, without inflammation	Contraceptive and anabolic steroids, antibiotics, HAART
Cholestatic hepatitis	Cholestasis with lobular necro inflammatory activity; may show bile duct destruction	Antibiotics, phenothiazine, statins
Hepatocellular necrosis	Spotty hepatocyte necrosis Massive necrosis Chronic hepatitis	Methyldopa, phenytoin Acetaminophen, halothane Isoniazid
Fatty liver disease	Large and small droplet fat	Ethanol, corticosteroids, methotrexate, total parenteral nutrition
	"Micro vesicular steatosis" (diffuse small droplet fat)	Valproate, tetracycline, [illegible] (Reye syndrome), HAART
	Steatohepatitis with Mallory-Denk bodies	Ethanol, amiodarone
Fibrosis and cirrhosis	Periportal and pericellular fibrosis	Alcohol, methotrexate, enalapril, vitamin A and other retinoids
Granulomas	Noncaseating epithelioid granulomas Fibrin ring granulomas	Sulfonamides, amiodarone, isoniazid Allopurinol
Vascular lesions	Sinusoidal obstruction syndrome (veno-occlusive disease): obliteration of central veins	High-dose chemotherapy, bush teas
	Budd-Chiari syndrome Peliosis hepatis: blood-filled cavities, not lined by endothelial cells	Oral contraceptives Anabolic steroids, tamoxifen
Neoplasms	Hepatocellular adenoma	Oral contraceptives, anabolic steroids
	Hepatocellular carcinoma	Alcohol, thorotrast
	Cholangiocarcinoma	Thorotrast
	Angiosarcoma	Thorotrast, vinyl chloride

668. B

669. B

Hepatocellular adenomas are benign tumors of neoplastic hepatocytes. Most can be subclassified on the basis of molecular changes:

- HNF1-α inactivated adenomas, with virtually no risk of malignant transformation, often associated with oral contraceptive pill use or in individuals with MODY-3
- β-Catenin activated adenoma, with mutations in the β-catenin gene leading to marked atypia and associated with a very high risk for malignant transformation
- Inflammatory adenomas, the hallmark of which is up-regulation of C-reactive protein and serum amyloid A (often derived from gp130 mutations); 10% of these have concomitant β-catenin activating mutations. Risk for malignant transformation is intermediate.

670. A

Ménétrier disease is a rare disorder associated with excessive secretion of transforming growth factor α (TGF-α). It is characterized by diffuse hyperplasia of the foveolar epithelium of the body and fundus and hypoproteinemia due to protein-losing enteropathy. Secondary symptoms, such as weight loss, diarrhea, and peripheral edema, are commonly present. Symptoms and pathologic features of Ménétrier disease in children are similar to those in adults, but pediatric

disease is usually self-limited and often follows a respiratory infection. Risk of gastric adenocarcinoma is increased in adults with Ménétrier disease

671. D

Neoplastic tubular, villous and tubulovillous adenomatous polyps are seen in FAP, tubular most commonly.

672. C

A is the name of a gene, CD 117 is the marker used on IHC and it is the most sensitive marker of GIST.

673. A

Lynch syndrome (Hereditary Non Polyposis Colon Cancer) due to defects in DNA Mismatch repair genes like MSH2 and MLH1

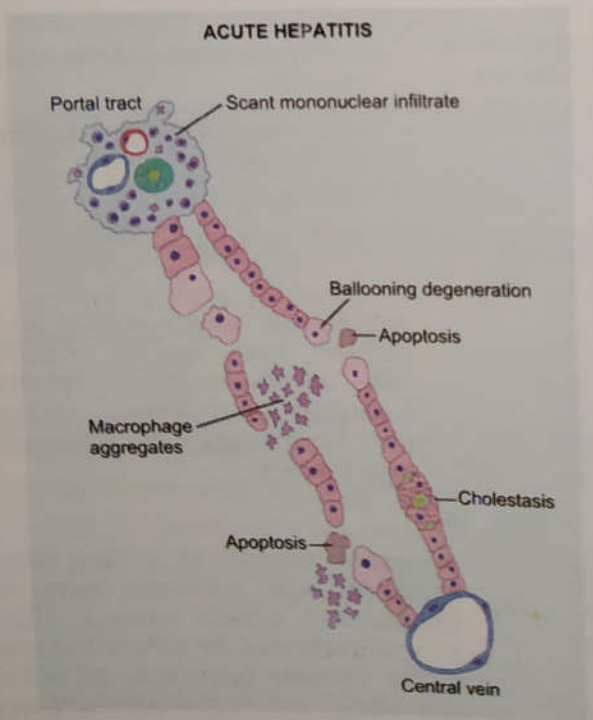
674. B

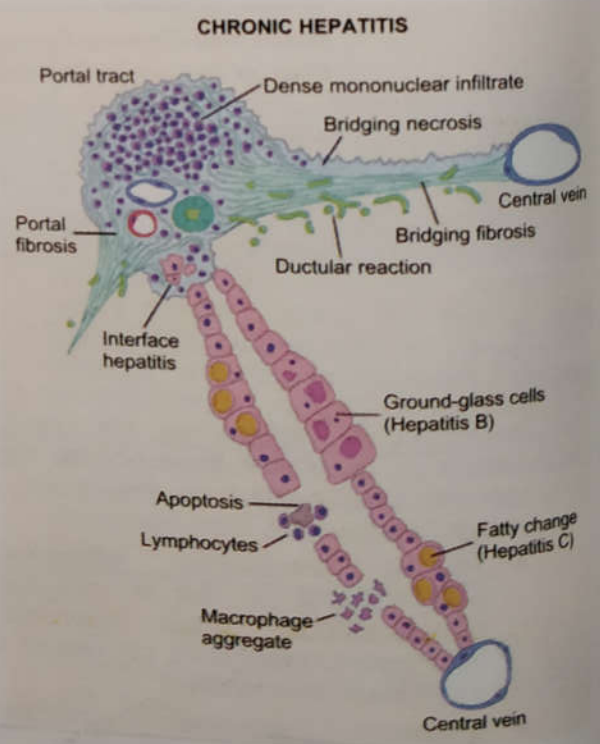
Malakoplakia is a distinctive chronic inflammatory reaction that appears to stem from acquired defects in phagocyte function, malakoplakia arises in the setting of chronic bacterial infection, mostly by E. coli or occasionally Proteus species. It occurs with increased frequency in immunosuppressed transplant recipients. In the bladder, malakoplakia takes the form of soft, yellow, slightly raised mucosal plaques, 3 to 4 cm in diameter, that are filled with large, foamy macrophages mixed with occasional multinucleate giant cells and lymphocytes. The macrophages have an abundant granular cytoplasm due to phagosomes stuffed with particulate and membranous debris of bacterial origin. In addition, laminated mineralized concretions resulting from deposition of calcium in enlarged lysosomes, known as Michaelis-Gutmann bodies, are

675. B

Also called chicken wire fibrosis

676. D





677. A

α1-Antitrypsin deficiency is an autosomal recessive dis-Antitrypsin (α1order of protein folding marked by very low levels of circulating α1AT). The major function ofthis protein is the inhibition of proteases, particularly neutrophil elastase, cathepsin G, and proteinase 3, which are normally released from

neutrophils at sites of inflammation. α1AT deficiency leads to the development of pulmonary emphysema, because the activity of destructive proteases is not inhibited. It also causes liver disease as a consequence of hepatocellular accumulation of the misfolded protein. Cutaneous necrotizing panniculits also occurs in a minor subset of patients. α1AT is a small 394–amino acid plasma glycoproteinsynthesized predominantly by hepatocytes. It is a member of the serine protease inhibitor (serpin) family. The gene, located on chromosome 14, is very polymorphic.

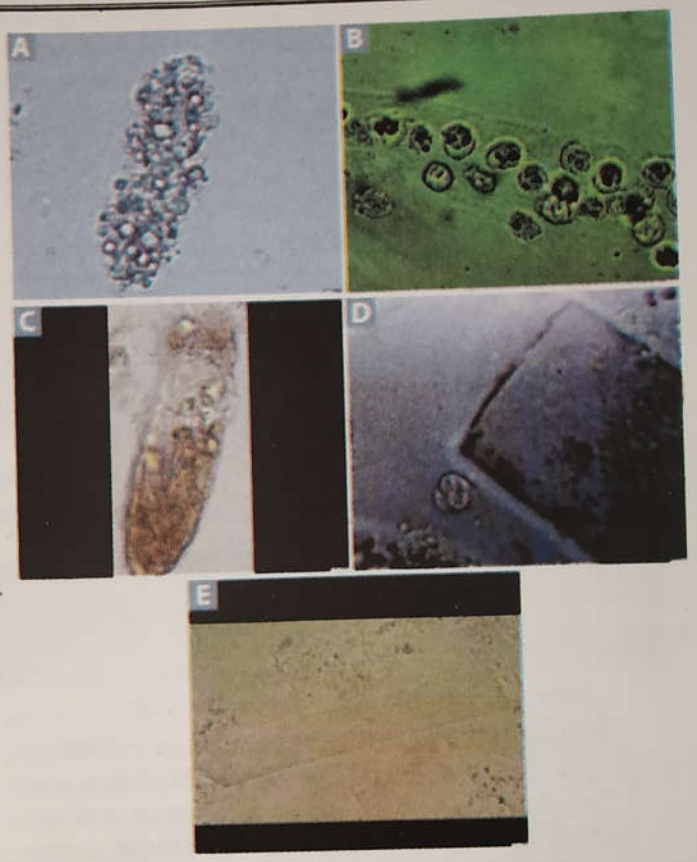
678. D

HFE gene located on chromosome 6p

679. A

As per latest update in the new edition of Harrison's Principles of Internal Medicine, KRAS is seen in almost 100% cases of pancreatic cancer.

680. A, B, C, E



RBC casts A	Glomerulonephritis, hypertensive emergency.
WBC casts B	Tubulointerstitial inflammation, acute pyelonephritis, transplant rejection.
Fatty casts ("oval fat bodies")	Nephrotic syndrome. Associated with "Maltese cross" sign.
Granular ("muddy brown") casts C	Acute tubular necrosis (ATN).
Waxy casts D	End-stage renal disease/chronic renal failure.
Hyaline casts E	Nonspecific, can be a normal finding, often seen in concentrated urine samples.

681. C

Option A- PSGN is a nephritic syndrome with non nephrotic range proteinuria
Option B- seen in acute pyelonephritis
Option D- seen in anti GBM antibody mediated disorder

682. C

Collapsing type of FSGS with visceral cell hypertrophy and retraction of glomerulus is classical of HIV.

683. A

Option B – seen in membranous nephropathy
Option C- seen in IgA nephropathy
Option D- seen in MPGN type I

684. A, B, C, D

Salient Features of Different Glomerulopathies

Disease	Clinically	Light Microscopy (H And E, Silver/Pas Special Stain)	Immunofluo-rescence	Electron Microscopy
Minimal change disease	Nephrotic	No change	Negative	Effacement of foot processes
Focal segmental glomerulosclerosis	Predominantly nephrotic	Focal and segmental hyalinosis and sclerosis	Usually negative	Effacement of foot processes, epithelial denudation

Membranous glomerulopathy	Nephrotic	Capillary thickening, "spike and dome" appearance	Granular IgG and C3	Subepithelial deposits
Membranoproliferative glomerulonephritis type 1	Nephrotic/ Nephritic	Mesangial and endocapillary proliferation, "tram track" appearance	Granular IgG, C3, Clq, C4	Subendothelial deposits
Acute infectious glomerulonephritis	Nephritic	Endocapillary proliferation, mesangial proliferation, leucocytic infiltration	Granular IgG, C3	Primarily subepithelial (early-subendothelial)
IgA nephropathy	Recurrent hematuria	Mesangial proliferation	IgA +/- IgG, IgM, C3 in the mesangium	Mesangial and paramesangial deposits
Pauci immune glomerulonephritis (ANCA mediated)	Rapidly proliferative glomerulonephritis	Extra capillary proliferation, crescent formation	Fibrin in crescents	No deposits, fragmented GBM

685. B

Also called Dense Deposit Disease

Most patients with dense-deposit disease (formerly called type II MPGN) have abnormalities resulting in excessive activation of the alternative complement pathway. These patients have a consistently decreased serum C3 but normal C1 and C4, the early components of complement. They also have diminished serum levels of Factor B and properdin, components of the alternative complement pathway. In the glomeruli, C3 and properdin are deposited, but IgG is not. Recall that in the alternative complement pathway, C3 is directly cleaved to C3b. The reaction depends on the initial activation of C3 by such substances as bacterial polysaccharides, endotoxin, and aggregates of IgA via a pathway involving Factors B and D. This leads to the generation of C3bBb, the alternative pathway C3 convertase. Normally, this C3 convertase is labile, but more than 70% of patients with dense-deposit disease have a circulating autoantibody termed C3 nephritic factor (C3NeF) that binds the alternative pathway C3 convertase and protects it from inactivation. This favors persistent C3 activation and hypocomplementemia.

686. B

IF shows granular pattern and EM shows subepithelial deposits hence suggestive of B.

687. C

688. A

History is suggestive of diabetic retinopathy and other related complications. The histopathology shows nodular glomerulosclerosis also known as Kimmelstein Wilson nodules.

689. B

Armanni-Ebstein lesions were first described by Luciano Armanni, a pathologist at the University of Naples, during autopsy studies undertaken in 1872, as a unique vacuolar nephropathy associated with poorly controlled diabetes that involves selective renal epithelial cell glycogen accumulation. However, within the last two decades, a broader range of vacuolar changes, including lipid deposition, have also been termed Armanni-Ebstein (AE) lesions, creating some confusion on possible etiology. The term AE phenomenon is best reserved for the original clear cell change associated with glycogen deposition, and that this should be clearly distinguished from subnuclear lipid vacuolization («basal vacuolization»).

690. B

The history is suggestive of malignant hypertension. The kidney size varies depending on the duration and severity of the hypertensive disease. Small, pinpoint petechial hemorrhages may appear on the cortical surface from rupture of arterioles

or glomerular capillaries, giving the kidney a peculiar "flea-bitten" appearance.

691. C

Most common type of RCC is clear cell type in which VHL gene mutations are most common.

692. D

Chromophobe carcinoma represents 5% of renal cell cancers and is composed of cells with prominent cell membranes and pale eosinophilic cytoplasm, usually with a halo around the nucleus. On cytogenetic examination these tumors show multiple chromosome losses and extreme hypodiploidy. Like the benign oncocytoma, they are thought to grow from intercalated cells of collecting ducts and have an excellent prognosis compared with that of the clear cell and papillary cancers. Histologic distinction from oncocytoma can be difficult.

typically present within the macrophages.

693. A

Disease	Inheritance	Pathologic Features	Clinical Features of Complications	Typical Outcome	Diagrammatic Representation
Adult polycystic kidney disease	Autosomal dominate	Large multicystic kidneys, liver cysts, berry aneurysms	Hematuria, flank pain, urinary tract infection, renal stones, hypertension	Chronic renal failure beginning at age 40-60 years	
Childhood polycystic kidney disease	Autosomal recessive	Enlarged, cystic kidneys at birth	Hepatic fibrosis	Variable, death in infancy of childhood	
Medullary sponge kidney	None	Medullary cysts on excretory urography	Hematuria, urinary tract infection, recurrent renal stones	Bening	
Familial juvenile nephronophthisis	Autosomal recessive	Corticomedullary cysts, shrunken kidneys	Salt wasting, polyuria, growth retardation, anemia	Progressive renal failure beginning in childhood	
Adult-onset medullary cystic disease	Autosomal dominate	Corticomedullary cysts, shrunken kidneys	Salt wasting, polyuria	Chronic renal failure beginning in adulthood	
Multicystic renal dysplasia	None	Irregular kidneys with cysts to variable size	Association with order renal anomalies	Renal failure if bilateral, surgically curable if unilateral	

Acquired renal cystic disease	None	Cystic degeneration in end-stage kidney disease	Hemorrhage, erythrocytosis Neoplasis	Dependence on dialysis	
Simple cysts	None	Single or multiple cysts in normal-sized kidneys	Microscopic hematuria	Bening	

694. B

Thyroidisation of tubules is seen in chronic pyelonephritis. The microscopic changes involve predominantly tubules and interstitium. The tubules show atrophy in some areas and hypertrophy or dilation in others. Dilated tubules with flattened epithelium may be filled with casts resembling thyroid colloid (thyroidization). There are varying degrees of chronic interstitial inflammation and fibrosis in the cortex and medulla.

695. C

Atherosclerotic plaques have three principal components: (1) smooth muscle cells, macrophages, and T cells; (2) extracellular matrix, including collagen, elastic fibers, and proteoglycans; and (3) intracellular and extracellular lipid. These components occur in varying proportions and configurations in different lesions. Typically, there is a superficial fibrous cap composed of smooth muscle cells and relatively dense collagen. Beneath and to the side of the cap (the "shoulder") is a more cellular area containing macrophages, T cells, and smooth muscle cells. Deep to the fibrous cap is a necrotic core, containing lipid (primarily cholesterol and cholesterol esters), debris from dead cells, foam cells (lipidladen macrophages and smooth muscle cells), fibrin, variably organized thrombus, and other plasma proteins; the cholesterol content is frequently present as crystalline aggregates that are washed out during routine tissue processing and leave behind only empty "clefts."

696. D

Mönckeberg medial sclerosis is characterized by calcification of the walls of muscular arteries, typically involving the internal elastic membrane. Persons older than age 50 are most commonly affected. The calcificationsdo not encroach on the vessel lumen and are usually not clinically significant.

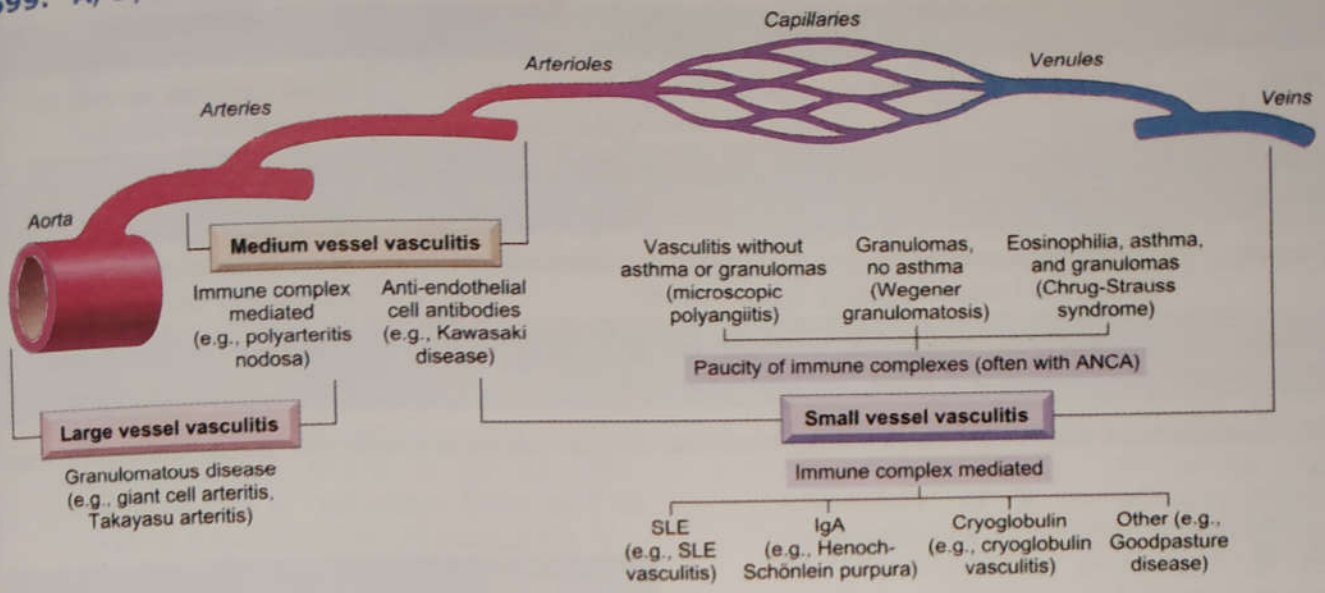
697. C

Hyaline arteriolosclerosis- arterioles show homogeneous, pink hyaline thickening with associated luminal narrowing. These changes reflect both plasma protein leakage across injured endothelial cells, as well as increased smooth muscle cell matrix synthesis in response to the chronic hemodynamic stresses of hyper tension. Although the vessels of older patients (either nor motensive or hypertensive) also frequently exhibit hyaline arteriosclerosis, it is more generalized and severe in patients with hypertension. The same lesions are also a common feature of diabetic microangiography; in that case, the underlying etiology is hyperglycemia-induced endothelial cell dysfunction. In nephrosclerosis due to chronic hypertension, the arteriolar narrowing of hyaline arteriosclerosis causes diffuse impairment of renal blood supply and glomerular scarring

698. B

Hyperplastic Arteriolosclerosis -this lesion occurs insevere hypertension; vessels exhibit concentric, laminated ("onion-skin") thickening of the walls with luminal narrowing.The laminations consist of smooth muscle cells with thickened, reduplicated basement membrane; in malignant hypertension, they are accompanied by fibrinoid deposits and vessel wall necrosis (necrotizing arteriolitis), particularly in the kidney.

699. A, D, E



700. D

	Giant Cell Arteritis	Granulomatosis with Polyangiitis	Churg-Strauss Syndrome	Polyarteritis Nodosa	Leukocytoclastic Vasculitis	Buerger Disease	Behcet Disease
Sites of Involvement							
Aorta	+	–	–	–	–	–	–
Medium-sized arteries	+	+	+	+	–	+	+
Small-sized arteries	–	+	+	+	+	+	+
Capillaries	–	–	-	-	+	–	+
Veins	–	–	–	–	+	+	+
Inflammatory Cells Present							
Lymphocytes	+	+	+	±	±	±	±
Macrophages	+	+	+	±	±	±	±
Neutrophils	Rare	+	+	±	±	±	Required
Eosinophils	Very rare	±	Required	±	±	±	±
Other Features							
Granulomas	±*	Required*	±	–	–	–	–
Giant cells	Often; not required	±	–	–	–	–	–
Thrombosis	±	±	±	±	±	Required	±
Serum ANCA positivity	–	+	+	+	–	–	–
Clinical history	>40 y years old, ± polymyalgia rheumatica	Any	Asthma, atopy	Any	Any	Young male smoker	Orogenital ulcers

*The granulomas of giant cell arteritis are found within the vessel wall as part of the inflammation comprising the vasculitis, but need not be present to render the diagnosis. The granulomas of granulomatosis with polyangiitis are larger, spanning between vessels, and associated with areas of tissue necrosis.

701. C

Type	Population	Clinical	Course
Classic	Older men (50-80 years)	Usually confined to lower extremity	Usually indolent, survival 10-15 years
Endemic (African)	Young black males, 15-40 yrs, and children	Localized nodular lesions or large exophytic, aggressive lesions	Nodules indolent; aggressive lesion survival 3-5 years
Iatrogenic	Immune suppressed (e.g., renal transplant)	Localized or widespread involvement	May regress when immune suppressants discontinued
Epidemic (AIDS-related)	Primarily gay men in U.S., Europe; adults in Africa	Head, face, neck, GI and lung most common	Fulminant, survival I-3 years without effective HIV therapy

AIDS, Amitendu Bhatt acquired immunodeficiency syndrome; GI, gastrointestinal system; HIV, human immunodeficiency virus.

702. D

Endothelial cell marker is PECAM/CD 31 seen in tumors of endothelial cell origin.

703. A

Classically, endocardium and valves of the right heart are primarily affected since they are the first cardiac tissues bathed by the mediators released by gastrointestinal carcinoid tumors. The cardiovascular lesions associated with the carcinoid syndrome are distinctive, glistening white intimal plaquelike thickenings of the endocardial surfaces of the cardiac chambers and valve leaflets

704. B

Chambers
Increased left atrial cavity size
Decreased left ventricular cavity size
Sigmoid-shaped ventricular septum
Valves
Aortic valve calcific deposits
Mitral valve annular calcific deposits
Fibrous thickening of leaflets
Buckling of mitral leaflets toward the left atrium
Lambl excrescences
Epicardial Coronary Arteries
Tortuosity
Diminished compliance
Calcific deposits
Atherosclerotic plaque
Myocardium
Decreased mass
Increased subepicardial fat
Brown atrophy
Lipofuscin deposition
Basophilic degeneration
Amyloid deposits
Aorta
Dilated ascending aorta with rightward shift
Elongated (tortuous) thoracic aorta
Sinotubular junction calcific deposits
Elastic fragmentation and collagen accumulation
Atherosclerotic plaque

705. C

This gross histochemical stain imparts a brick-red color to intact, noninfarcted myocardium where lactate dehydrogenase activity is preserved. Because dehydrogenases leak out through the damaged membranes of dead cells, an infarct appears as an unstained pale zone.

706. C

Regurgitant jets due to mitral valve dysfunction leading to plaque formation in the posterior wall in the sub endothelium of left atrium are called Mac Callum patches seen in rheumatic heart disease.

707. B

Fatty degenerated heart in which the fat is disposed in the form of broken stripes

in the subendocardial myocardium is called tigered effect.

708. B

Lobular breast carcinoma has a tendency to be multifocal and bilateral, hence contralateral breast should always be examined

709. D

Medullary carcinoma is of great interest due to the findingthat many tumors of this type have features that are characteristic of BRCA1associated carcinomas. Among cancers arising in BRCA1 carriers, 13% are of medullary type, and up to 60% have a subset of medullary features. Although the majority of medullary carcinomas are not associated with germline BRCA1 mutations, hypermethylation of the BRCA1 promoter leading to downregulation of BRCA1 expression is observed in 67% of these tumors. The basis for the relatively good prognosis of this subtype compared to other poorly differentiated carcinomas is not known, but it has been noted that the presence of lymphocytic infiltrates within the tumors is associated with higher survival rates and a greater response to chemotherapy, suggesting that improved outcomes may be related to a host immune response to tumor antigens. Medullary carcinoma is softer than other carcinomas (medulla is Latin for "marrow") due to minimal desmoplasia, and often presents as a well-circumscribed mass. It is characterized by (1) solid, syncytium-like sheets of large cells with pleomorphic nuclei, and prominent nucleoli, which compose more than 75% of the tumor mass; (2) frequent mitotic figures; (3) a moderate to marked lymphoplasmacytic infiltrate surrounding and within the tumor; and (4) a pushing (noninfiltrative) border. DCIS is minimal or absent

710. B

Approximately 12% of breast cancers occur due to inheritance of an identifiable susceptibility gene or genes. The probability of a hereditary etiology increases when there are multiple affected firstdegree relatives, early onset cancers, multiple cancers, or family members with other specific cancers.Mutations in BRCA1 and BRCA2 are responsible for80% to 90% of "single gene" familial breast cancers and about 3% of all breast cancers. Penetrance (the percentage of carriers who develop breast cancer) varies from 30% to 90% depending on the specific mutation present. Mutations in BRCA1 also markedly increase the risk of developing ovarian carcinoma, which occurs in as many as 20% to 40% of carriers. BRCA2 confers a smaller risk for ovarian carcinoma (10% to 20%) but is associated more frequently with male breast cancer. BRCA1 and BRCA2 carriers are also athigher risk for other epithelial cancers, such as prostatic and pancreatic carcinomas.

711. B

Feature	ER Positive / HER2 Negative	HER2 Positive (ER Positive or Nagative)	Triple Negative (ER, PR, and HER2 Negative)
Overall frequency	50%-65%	20%	15%
Typical patient groups	Older women; men; cancers detected by screening; germline BRCA2 mutation carries	Young women; germline TP53 mutation carries	Young women; germline BRCA / mutation carries
Ethnicity			
European/American	70%	18%	12%
African/American	52%	22%	26%

Hispanic	60%	24%	16%
Asian/ Pacific Islander	63%	26%	11%
Grade	Mainly grade 1 and 2	Mainly grade 2 and 3	Mainly grade 3
Complete response to chemotherapy	Lower grade (<10%), higher grade (10%)	ER positive (15%), ER negative (>30%)	30%
Timing of relapse	May be late (>10 years after diagnosis)	Usually short (<10 years after diagnosis)	Usually short (<8 years after diagnosis
Metastatic sites	Bone (70%), viscera (25%), brain (<10%)	Bone (70%), viscera (45%), brain (30%)	Bone (40%), viscera (35%), brain (25%)
Similar group defined by Mrna profiling	Luminal A (low grade), luminal B (high grade)	Luminal B (ER positive), HER2-enriched (ER negative)	Basal-like
Common special histologic types	Lobular, tubular, mucinous, papillary	Apocrine, micropapilary	Carcinoma with medullary features
Common somatic mutations	PIK3CA (40%), TP53 (26%)	TP53 (75%), PIK3CA (40%)	TP53 (85%)

712. B

713. A

714. B

Option C- Most important prognostic factor for metastatic breast cancer

715. B

Lobular carcinoma most commonly forms hard irregular masses similar to other breast cancers, but may also have a diffuse infiltrative pattern with minimal desmoplasia. Such cancers can be difficult to palpate or detect by imaging. This is the most common type of breast carcinoma to present as an occult primary. The histologic hallmark is the presence of discohesive infiltrating tumor cells, often including signet-ring cells containing intracytoplasmic mucin droplets. Tubule formation is absent.

716. B

Inflammatory breast cancers show extensive invasion and proliferation within lymphatic channels, causing swelling that mimics non-neoplastic inflammatory lesions. These tumors are usually of high grade, but do not belong to any particular molecular subtype.

717. C

Pathologic Lesion	Relative Risk (Absolute Lifetime Risk)*
Nonproliferative Breast Changes (Fibrocystic changes)	1 (3%)
Duct ectasia	
Cysts	
Apocrine change	
Mild hyperplasia	
Adenosis	
Fibroadenoma without complex features	

Proliferative Disease Without Atypia	1.5 to 2 (5%-7%)
Moderate or florid hyperplasia Sclerosing adenosis Papilloma Complex sclerosing lesion (radial scar) Fibroadenoma with complex features	
Proliferative Disease with Atypia	4 to 5 (13%-17%)
Atypical ductal hyperplasia (ADH) Atypical lobular hyperplasia (ALH)	
Carcinoma in Situ	8 to 10 (25%-30%)
Lobular carcinoma in situ (LCIS) Ductal carcinoma in situ (DCIS)	

Relative risk is the risk compared to women without any risk factors. Absolute lifetime risk is the percentage of patients expected to develop invasive carcinoma if untreated.

718. C

Adenocarcinomas of GIT, Lung, Breast and Pancreas are associated with migratory thrombophlebitis.

719. A

The progression of the dementia in CJD is usually so rapid that there is little if any grossly evident brain atrophy. The pathognomonic finding is a spongiform transformation of the cerebral cortex and, often, deep gray matter structures (caudate, putamen); this multifocal process results in the uneven formation of small, apparently empty, microscopic vacuoles of varying sizes within the neuropil and sometimes in the perikaryon of neurons

720. D

Schwannomas are well-circumscribed, encapsulated masses that abut the associated nerve without invading it, a feature that simplifies surgical excision. Grossly, these tumors form firm, gray masses. Microscopically, they are comprised of an admixture of dense and loose areas referred to as Antoni A and Antoni B areas, respectively. The dense eosinophilic Antoni A areas often contain spindle cells arranged into cellular intersecting fascicles. Palisading of nuclei is common and "nuclear-free zones" that lie between the regions of nuclear palisading are termed Verocay bodies. In the loose, hypocellular Antoni B areas the spindle cells are spread apart by a prominent myxoid extracellular matrix that may be associated with microcyst formation.

721. A

Homologous recombination repair gene defect – Bloom syndrome, Fanconi Anemia, and Ataxia telangiectasia

722. B

723. B

Option A- mesenchymal tumor origin
Option C and D- muscle tumor origin

724. C

Tumor	Cytogenetic Abnormality	Gene fusion
Ewing sarcoma family tumors	t (11 ;22) (q24;q12)	EWS-FLI1
	t (21 ;22) (q22;q12	EWS-ERG
Extraskeletal myxoid chondrosarcoma	t (9;22) (q22;q12)	EWS-CHN
Desmoplastic small round-cell tumor	t (11 ;22) (p13;q12)	EWS-WT1
Clear-cell sarcoma	t (12;22) (q13;q12)	EWS-ATF1

Liposarcoma–myxoid and round-cell type	t (12;16) (q13;p11)	FUS-DDIT3
Synovial sarcoma	t (x;18) (p11;q11)	SS18-SSX1 SS18-SSX2 SS18-SSX4
Rhabdomyosarcoma–alveolar type	t (2;13) (q35;q14) t (1;13) (p36;q14)	PAX3-F0X01 PAX7-F0X01
Dermatofibrosarcoma protuberans	t (17;22) (q22;q15)	COLA1-PDGFB
Alveolar soft-part sarcoma	t (X;17) (p11.2;q25)	TFE3-ASPL
Infantile fibrosarcoma	t (12;15) (p13;q23)	ETV6-NTRK3
Nodular fasciitis	t (22;17)	MYH9-USP6

725. C

Description is of Schiller Duval bodies in Yolk sac or endodermal sinus tumor

726. A

727. A

Option B- most common cause of nephrotic syndrome in children

728. B

HIV encephalitis is a chronic inflammatory reaction associated with widely distributed microglial nodules, often containing macrophage-derived multinucleated giant cells; foci of tissue necrosis and reactive gliosis are sometimes seen together with these lesions

729. A

Option B- most common primary CNS malignancy in children

730. C

Elongated cells are referred to as strap cells or tadpole cells. Rhabdomyoblasts contain an eosinophilic granular cytoplasm rich in thick and thin filaments. The presence of desminand myogenin can be detected using immunohistochemistry.

731. A

Familial adenomatous polyposis is an inherited condition with neoplastic polyps, whereas JPS and PJP are classified under non neoplastic hamartomatous polyps. Ulcerative colitis and Crohn's disease comprise the spectrum of inflammatory bowel disease and therefore also carry a risk of colorectal cancer (equal incidence).

732. C

The location, size and electron microscopy picture of this lesion goes in favour of a carcinoid tumor or a neuroendocrine tumor. The best marker to identify such lesions is synaptophysin and chromogranin.

733. C

The IHC pictures show nuclear positivity for ER and no membranous positivity for Her 2 Neu. This falls under the Luminal A group of molecular classification and has the best prognosis among all the subtypes. Since Her 2 Neu is negative, Trastuzumab is not an option for treatment but since ER is positive, hormonal therapy can be used. Basal subtype is triple negative, having one of the worst prognosis of all the tumors.

734. C

Since the history is short, the possibility of acquiring IgA nephropathy is higher. It is characterised by mesangial deposits.

735. D

ALK is rearranged, not mutated in lung cancer. It is translocated leading to fusion of EML4-ALK.

736. C

Lymphoid follicles along with macrovesicular steatosis are seen in chronic hepatitis C

737. B

Most common carcinoma occurring in a patient with asbestos exposure is lung carcinoma and most specific is mesothelioma

738. D

Chromosomal aneuploidy is seen in malignancies

739. B

Cryptitis and crypt abscesses maybe be seen in both. Granuloma formation and transmural inflammation are characteristic of CD while pseudopolyposis is characteristic of UC.

740. B

Red pulp involving sinuses are the site of amyloid deposition in lardaceous spleen

741. A

CEA or carcinoembryonic antigen is seen in both colon and pancreatic cancers and other adenocarcinomas as well. CA 125 is a marker for ovarian carcinoma and CA 15-3 is a marker for breast carcinoma.

742. D

Most common primary tumors metastasising to brain are lung, breast, melanoma, kidney and GIT. Prostate carcinoma has a tendency to primarily involve the bones, not brain.

743. C

A longitudinal groove in the nucleus is seen in tumor cells of Brenner tumor, Granulosa cell tumor, LCH, Chondroblastoma and papillary carcinoma thyroid.

744. C

Bone invasion does not upgrade meningioma. Chordoid and clear cell morphology is grade II and rhabdoid and papillary morphology is grade III. EMA and PR are expressed.

745. C

Triphenyl tetrazolium chloride is a gross histochemical stain which impartsa brick-red color to intact, noninfarcted myocardium where lactate dehydrogenase activity is preserved. Because dehydrogenases leak out through the damaged membranes of dead cells, an infarct appears as an unstained pale zone By 12 to 24 hours after infarction, an MI can usually be identified grossly as a reddish-blue area of discoloration caused by stagnated, trapped blood. progressively more sharply defined, yellow-tan, and soft. By 10 days to 2 weeks, it is rimmed by a hyperemic zone of highly vascularized granulation tissue. Over the succeeding weeks, the injured region evolves to a fibrous scar.

746. A

The skin, mucous membranes, lungs, brain, heart, gastrointestinal tract, kidneys, and muscle can all be involved; necrotizing glomerulonephritis (90% of patients) and pulmonary capillaritis are particularly common. Microscopic angiitis can be a feature of a number of immune disorders, such as Henoch-Schönlein purpura, essential mixed cryoglobulinemia, and vasculitis associated with connective tissue disorders

747. C

A velvety patch in lower esophagus with the clinical history indicative of reflux suggests Barret's esophagus. Option B is the histopathological picture best describing the condition. Option A is the description for squamous cell carcinoma. Option B is the description for adenocarcinoma.

748. C

The peripheral smear shows acanthocytes and the intestinal biopsy shows vacuolated epithelial cells suggestive of abetalipoproteinemia.

749. C

Medullary carcinomas of the thyroid are neuroendocrine neoplasms derived from the parafollicular cells, or C cells, of the thyroid, and account for approximately 5% ofthyroid neoplasms. Medullary carcinomas, similar tonormal C cells, secrete *calcitonin*, the measurement of whichplays an important role in the diagnosis and postoperativefollow-up of patients. In some instances the tumor cellselaborate other poly-

peptide hormones, such as serotonin, ACTH, and vasoactive intestinal peptide (VIP). About 70%of tumors arise sporadically. The remainder occurs in the setting of MEN syndrome 2A or 2B or as familial tumorswithout an associated MEN syndrome.

750. A Capillary hemangioma

Capillary hemangiomas are the most common type; these occur in the skin, subcutaneous tissues, and mucous membranes of the oral cavities and lips, as well as in the liver, spleen, and kidneys. Histologically, they are composed of thin-walled capillaries with scant stroma

751. D. APC

The photograph shows numerous adenomas of the colon, consistent with familial adenomatous polyposis (FAP), also termed adenomatous polyposis coli (APC). This autosomal dominant inherited disease accounts for about 1% of colorectal cancers. It is characterized by the progressive development of innumerable adenomatous polyps of the colorectum, particularly in the rectosigmoid region.

Germline mutations in the APC gene, a putative tumor suppressor gene, are responsible for FAP. Carcinoma of the colon and rectum is inevitable in these patients, and the mean age of onset is 40 years

752. B. S-100 is negative

S-100 is positive in nerve sheath tumors like schwannomas. Rest of the statements are true.

753. C. Intestinal wall is thinned out

Feature	Crohn Disease	Ulcerative Colitis
Macroscopic		
Bowel region affected	Ileum ± colon	Colon only
Rectal involvement	Sometimes	Always
Distribution	Skip lesions	Diffuse
Stricture	Yes	Rare
Bowel wall appearance	Thick	Thin
Inflammation	Transmural	Limited to mucosa and submucosa
Pseudopolyps	Moderate	Marked
Ulcers	Deep, knifelike	Superficial, broad-based
Lymphoid reaction	Marked	Moderate
Fibrosis	Marked	Mild to none
Serositis	Marked	No
Granulomas	Yes (-35%)	No
Fistulas/sinuses	Yes	No
Clinical		
Perianal fistula	Yes (in colonic disease)	No
Fat/vitamin malabsorption	Yes	No
Malignant potential	With colonic involvement	Yes
Recurrence after surgery	Common	No
Toxic megacolon	No	Yes

754. A. Cytokeratin 8/18

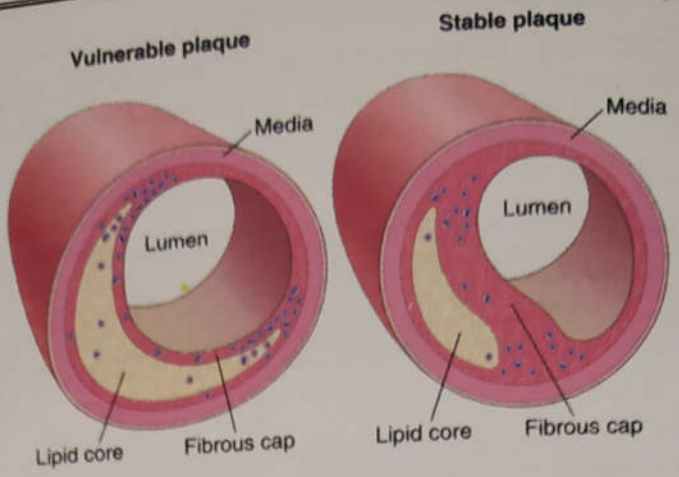
MalloryDenkbodiesconsist of tangledskeins of intermediatefilaments (including ubiquitinylatedkeratins 8 and 18) and arevisible as eosinophiliccytoplasmicinclusions in degeneratinghepatocytes

755. A. Luminal A subtype

Feature	ER Positive / HER2 Negative	HER2 Positive (ER Positive or Nagative)	Triple Negative (ER, PR, and HER2 Negative)
Overall frequency	50%-65%	20%	15%
Typical patient groups	Older women; men; cancers detected by screening; germline BRCA2 mutation carries	Young women; germline TP53 mutation carries	Young women; germline BRCA / ... ...rries
Ethnicity			
European/American	70%	18%	12%
African/American	52%	22%	26%
Hispanic	60%	24%	16%
Asian/ Pacific Islander	63%	26%	11%
Grade	Mainly grade 1 and 2	Mainly grade 2 and 3	Mainly grade 3
Complete response to chemotherapy	Lower grade (<10%), higher grade (10%)	ER positive (15%), ER negative (>30%)	30%
Timing of relapse	May be late (>10 years after diagnosis)	Usually short (<10 years after diagnosis)	Usually short (<8 years after diagnosis
Metastatic sites	Bone (70%), viscera (25%), brain (<10%)	Bone (70%), viscera (45%), brain (30%)	Bone (40%), viscera (35%), brain (25%)
Similar group defined by Mrna profiling	Luminal A (low grade), luminal B (high grade)	Luminal B (ER positive), HER2-enriched (ER negative)	Basal-like
Common special histologic types	Lobular, tubular, mucinous, papillary	Apocrine, micropapilary	Carcinoma with medullary features
Common somatic mutations	PIK3CA (40%), TP53 (26%)	TP53 (75%), PIK3CA (40%)	TP53 (85%)

756. A

Stable plaques have densely collagenized and thickened fibrous caps with minimal inflammation and negligible underlying atheromatous cores, whereas vulnerable plaques have thin fibrous caps, large lipid cores, and increased inflammation.



757. D

One "traditional" 10% rule that has since been modified pertains to familial cases. It is now recognized that as many as 25% of individuals with pheochromocytomas and paragangliomas harbor a germ line mutation in one of at least six known genes, including RET, which causes type 2 MEN syndromes; NF1, which causes type 1 neurofibromatosis ; VHL, which causes von Hippel Lindau disease; and three genes encoding subunits within the succinate dehydrogenase complex (SDHB, SDHC, and SDHD), which is involved in mitochondrial electron transport and oxygen sensing. It is postulated that loss of function in one or more of these subunits leads to stabilization of the transcription factor hypoxia-inducible factor 1α (HIF-1α), promoting tumorigenesis

758. A

The most frequent pituitary tumor in patients with MEN-1 syndrome is a prolactin-secreting macroadenoma.

759. C

Who histological classification of gastric tumours1

Epithelial tumours		Non-epithelial tumours	
Intraepithelial neoplasia - Adenoma	8140/0	Leiomyoma	8890/0
Carcinoma		Schwannoma	9560/0
Adenocarcinoma	8140/3	Granular cell tumour	9580/0
diffuse type	8144/3	Glomus tumour	8711/0
Papillary adenocarcinoma	8145/3	Leiomyosarcoma	8890/0
Tubular adenocarcinoma	8211/3	GI Stromal tumour	8936/1
Mucinous adenocarcinoma	8480/3	benign	8936/0
Signet-ring cell carcinoma	8490/3	uncertain malignant potential	8936/1
Adenosquamous carcinoma	8560/3	malignant	8936/3
Squamous cell carcinoma	8070/3	Kaposi sarcoma	9140/3
Small cell carcinoma	8041/3	Others	
Undifferentiated carcinoma	8020/3	Malignant lymphomas	
Others		Marginal zone B-cell lymphoma of MALT-type	9699/3
Carcinoid (well differentiated endocrine neoplasm)	8240/3	Mantle cell lymphoma	9673/3
		Diffuse large B-cell lymphoma	9680/3
		Others	
		Secondary tumours	

760. B

Myxomas are the most common primary tumor of the adult heart. These are benign neoplasms thought to arise from primitive multipotent mesenchymal cells. Although sporadic myxomas do not show consistent genetic alterations, familial syndromes associated with myxomas have activating mutations in the GNAS1 gene, encoding a subunit of G protein (Gsα) (in association with McCune-Albright syndrome) or null mutations in PRKAR1A, encoding a regulatory subunit of a cyclic-AMPdependent protein kinase (Carney complex). About 90% of myxomas arise in the atria, with a left-to-right ratio of approximately 4 : 1.

761. A

Multiple immunologic mechanisms that may contribute to thyroid cell damage-have been identified including the following:

- CD8+ cytotoxic T-cell–mediated killing of thyroid epithelial cells.
- Cytokine-mediated cell death. T-cell activation leads to the production of inflammatory cytokines such as interferon-γ in the thyroid gland, with resultant recruitment and activation of macrophages and damage to follicles.
- Binding of anti-thyroid antibodies (anti-thyroglobulin, and anti-thyroid peroxidase antibodies), followed by antibody-dependent cell–mediated cytotoxicity

In the usual clinical course, hypothyroidism develops gradually. In some cases, however, it may be preceded by transient thyrotoxicosis caused by disruption of thyroid follicles, with secondary release of thyroid hormones (hashitoxicosis). During this phase, free T4 and T3 concentrations are elevated, TSH is and T3 diminished, and radioactive iodine uptake is decreased. As hypothyroidism supervenes, T4 levels progressively fall, accompanied by a compensatory increase in TSH.

762. A

Involvement of the liver by metastatic malignancy is far more common than primary hepatic neoplasia. Although the most common primary sources are the colon, breast, lung, and pancreas, any cancer in any site of the body may spread to the liver. Typically, multiple nodular metastases are found that often cause striking hepatomegaly and replace much of the normal liver parenchyma. The liver weight can exceed several kilograms.

763. A, B, E **764. A**

765. A **766. B**

767. B

768. C (single best answer)

769. C **770. C**

771. C

772. A All are associated.

773. A

774. A (single best option)

775. C **776. D**

777. D **778. B**

779. C **780. B**

781. CD **782. C**

783. A, B, C, E **784. A**

785. D **786. A**

787. C **788. C**

789. D **790. C**

791. B **792. B**

793. B **794. A**

795. B **796. D**

797. D **798. A**

799. B **800. B**

801. D **802. B**

803. C **804. D**

805. A **806. B**

807. A **808. C**

809. D

810. B. Mucoepidermoid carcinoma

Mucoepidermoid carcinomas are composed of variable mixtures of squamous cells, mucus-secreting cells, and intermediate cells. These neoplasms represent about 15% of all salivary gland tumors, and while they occur mainly (60%–70%) in the parotids, they account for a large fraction of salivary gland neoplasms in the other glands, particularly the minor salivary glands. Overall, mucoepidermoid carcinoma is the most common form of primary malignant tumor of the salivary glands

811. B **812.** D
813. D **814.** D
815. D **816.** A, E
817. D **818.** A
819. A **820.** A
821. C **822.** D
823. D **824.** A
825. A **826.** A
827. B **828.** A
829. D **830.** A
831. B **832.** B
833. C **834.** C
835. A **836.** C
837. C **838.** D
839. C **840.** B

841. A

Option B- most common primary cardiac malignancy in children

842. C **843.** B

844. A, C, E

Lung adenocarcinoma is negative for CK 20 and CK 5/6
CK 5/6 is positive in mesothelioma

845. A

Increase in the thickness of mucus gland layer can cause increase in Reid's index.

846. A

Option B- Goodpasture syndrome
Option C- IgA nephropathy

847. A

FAP is due to APC gene mutations, present on chromosome 5.

848. B

Thrombangitis obliterans is caused due to smoking in young males in peripheral vessels.

849. D **850.** A
851. A **852.** A
853. C

854. B

ATP7B on chromosome 13q

855. B **856.** A
857. C **858.** B
859. D **860.** B
861. B

862. A

All types of invasive carcinoma are graded using the Nottingham Histologic Score. Carcinomas are scored for tubule formation, nuclear pleomorphism, and mitotic rate and the points added to divide carcinomas into grade I (well differentiated), grade II (moderately differentiated), and grade III (poorly differentiated) types.

863. D

Giant cell arteritis is a medical emergency requiring prompt recognition and treatment. Lesions also occur in other arteries, including the aorta (giant cell aortoarteritis)

864. C

865. A